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THE RELATION OF "CHRONIC MASTITIS" TO CARCINOMA OF THE BREAST

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DOES a given woman whose breast shows evidence of changes commonly described as chronic mastitis have greater chance of developing carcinoma than one with normal breasts? The problem which faces us is the importance of cystic disease as an etiological factor in carcinoma of the breast. Is the probability of a breast with chronic mastitis developing carcinoma sufficiently great to warrant simple mastectomy or even bilateral mastectomy; is it sufficiently slight to discard all fear and all caution or must a middle ground be taken? We have yet to find an authoritative opinion on this problem which so frequently confronts both surgeon and pathologist. This question would seem an easy one to answer and indeed many believe that they have solved it.

The present study was undertaken in an effort to present at least a partial answer in the hope that it might lead to a more complete solution ultimately. For this purpose a group of women, who had had portions of breast removed as a result of various symptoms, were followed for at least 5 years and the incidence of carcinoma was compared with that of the general female population.

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In view of the many terms applied to the various lesions of the breast, a very brief historical review may be helpful in orientation.

HISTORICAL REVIEW

Apparently the first association of cystic disease of the breast and carcinoma was made by Astley Cooper, who in 1845 in his *The Anatomy and Diseases of the Breast* records their coexistence.[1] While Brodie considered mammary cysts benign, he pointed out that they might recur, and that their presence does not rule out carcinoma. Billroth regarded the cysts as essentially the result of retention. Reclus considered the cysts to be neoplastic, but benign, although he admitted the possibility of the development of carcinoma from them in older women. He emphasized the bilateral character of the lesion. Careful study of the cysts led Brissaud to believe proliferation of the glandular epithelium the major factor in their formation. Schimmelbusch believed the cystic condition to be a true tumor and gave it the name of "cystadenoma mammæ." In 3 cases of 43 Schimmelbusch found carcinoma. Three cases of carcinoma developing in cystic breasts were also reported by Sicre. The term "chronic cystic mastitis" was first applied by Koenig, who believed the process to be inflammatory and

[1] Cited by Cheatle and Cutler.

TABLE I.—THE RELATION OF FIBRO-ADENOMATOSIS MAMMÆ TO THE DIFFERENT DEGREES OF CANCER DEVELOPMENT (GROUPS I TO IV). (FROM SEMB)

	Fibro-adenomatosis simplex (microcystica)				Fibro-adenomatosis cystica (simplex)				Fibro-adenomatosis cystica papillomatosa				Fibro-adenomatosis cystica (simplex +papillomatosa)			
	No. of cases	%	Average age	Bilateral preparation*	No. of cases	%	Average age	Bilateral preparation*	No. of cases	%	Average age	Bilateral preparation*	No. of cases	%	Average age	Bilateral preparation*
Group I, Fibro-adenomatosis cystica without infiltrating growth (i.e., without signs of carcinoma)	42	96	33 yrs.	2	42	57.5	42 yrs.	5	10	37	46 yrs.	0	52	52	43 yrs.	5
Group II, Fibro-adenomatosis cystica suspected of incipient infiltrating growth (i.e., suspected of carcinoma incipiens)	1	2	42 yrs.	0	14	19	44 yrs.	0	10	37	44.5 yrs.	2	24	24	44 yrs.	2
Group III, Fibro-adenomatosis cystica with incipient infiltrating growth (i.e., with carcinoma incipiens)	0	0		0	9	12.5	43 yrs.	0	5	18.5	45 yrs.	1	14	14	43.5 yrs.	1
Group IV, Fibro-adenomatosis cystica with pronounced infiltrating growth (i.e., with carcinoma mammæ)	1	2	38 yrs.	0	8	11	49.5 yrs.	0	2	7.5	48 yrs.	0	10	10	49 yrs.	0
Total cases	44	100	33 yrs.	2	73	100	43.5 yrs.	5	27	100	45.5 yrs.	3	100	100	44 yrs.	8

*Carcinoma on the other side and "clinical bilateralness" not included.

considered the epithelial proliferations as distinct from the cystic process. The epithelial proliferations within the cysts were emphasized by Tietze, who found cancer in 10 per cent of his cases. Ten per cent of Greenough and Hartwell's (16) 30 cases of cystic disease showed carcinoma. This change, named "adenomatous proliferation" by Warren, coexisted with carcinoma in 15 of 507 cases of breast cancer he reported. In Bloodgood's (5) first paper (1906) he called the condition "senile parenchymatous hypertrophy." He at that time regarded the adenocystic form as precancerous although he subsequently altered his opinion and finally considered carcinoma to develop no more frequently in this lesion than in the normal breast.

The belief in the precancerous nature of cystic disease was carried further by von Saar in 1907 (31), who believed the cystadenoma to be merely a phase in a physiological involution which finally ended in carcinoma.

Among 35 cases of "abnormal involution" Speese found 9 cases with carcinoma. Including his cases with a group from the literature, he found an association of carcinoma with abnormal involution in 15 per cent of 295 cases. According to Bertels the dilatation cysts were caused by separation of the acini by connective tissue hyperplasia. Following this cyst formation, transition to carcinoma might occur fairly frequently although only a small proportion of the mammary carcinomas developed from chronic cystic mastitis.

Greenough and Simmons (17) found an incidence of 4.8 per cent of carcinoma developing 1 to 17 years after excision of diseased foci from breasts the site of cystic disease.

Chronic cystic mastitis was regarded by Deaver and McFarland as abnormal involution. They recognized that the large, pale epithelial cells regarded by some (20, 32) as of sweat gland origin were produced by abnormal involutional changes. In examining 575 breasts removed for tumor, 335 of them cancerous, they encountered 23 in which abnormal involution and cancer coexisted. They concluded the proliferative and ectatic forms of abnormal involution to be benign, the adenocystic to develop cancer in about 10 per cent.

"Polycystic mammary gland degeneration," as Pribram termed the condition, may, in his opinion, be a precursor of carcinoma by irregular involutionary changes in the epithelium.

Krompecher (21) considered these cases in which there was a pronounced papillary ingrowth of epithelium and pale cells to be neoplastic and derived from sweat glands (hydrocystoma).

TABLE II.—ASSOCIATION OF FIBRO-ADENOMATOSIS CYSTICA WITH CANCER (FROM SEMB)

Group I. Fibro-adenomatosis cystica—without signs of infiltrating growth:		
a. Fibro-adenomatosis cystica simplex	43 cases out of	73
b. Fibro-adenomatosis cystica papillomatosa	10 cases out of	27
Total	53 cases out of	100
Group II. Fibro-adenomatosis cystica, suspected of incipient infiltrating growth (suspected of carcinoma incipiens):		
a. Fibro-adenomatosis cystica simplex	14 cases out of	73
b. Fibro-adenomatosis cystica papillomatosa	10 cases out of	27
Total	24 cases out of	100
Group III. Fibro-adenomatosis cystica with incipient infiltrating growth (with carcinoma incipiens):		
a. Fibro-adenomatosis cystica simplex	9 (8) cases out of	73
b. Fibro-adenomatosis cystica papillomatosa	5 cases out of	27
Total	14 (13) cases out of	100
Group IV. Fibro-adenomatosis cystica with pronounced infiltrating growth (with developed carcinoma):		
a. Fibro-adenomatosis cystica simplex	8 cases out of	73
b. Fibro-adenomatosis cystica papillomatosa	2 cases out of	27
Total	10 cases out of	100

One of the most careful studies is that of Semb, who analyzed a series of 144 cases of fibro-adenomatosis with coexistent cancer in a few. In 122 cases of carcinoma there were fibro-adenomas as well. His findings are summarized in Tables I and II. Carcinoma was present in 24 per cent of the group showing definite hyperplasia. On the basis of these he concludes: "The greatest importance must be attached to the relation to cancer."

McGlannan cited 3 instances of carcinoma of the breast coexistent with blue-domed cysts in a series of 100 breast cancers, and 5 showing cancer developing in a papillomatous cyst.

Cheatle and Cutler hold that 20 per cent of the cases of breast carcinoma arise on the basis of "cystipherous desquamative epithelial hyperplasia." They give no figures, however. In discussing a paper by Whitehouse on chronic mastitis, Cutler states that "at least 20 per cent of carcinomas have passed through the cystic and papillomatous changes."

Dietrich believes the breast in chronic cystic mastitis to be chronically inflamed as a result of abnormal involution. Sixty-eight, or 30 per cent, of 233 cases of chronic cystic mastitis showed coexistent cancer.

TABLE III.—TORONTO CASES BY YEARS, SHOWING INCIDENCE OF CANCER FOLLOWING DIFFERENT LESIONS

Year	Chronic cystic mastitis		Chronic mastitis		Duct papilloma	
	Total	Developed cancer	Total	Developed cancer	Total	Developed cancer
1920	16	1	6		1	
1921	19		10		3	
1922	24		8	1	1	
1923	31	2	14		4	
1924	18		9		6	
1925	28		13	1	5	
1926	36		13		5	
1927	41	1	12		11	
1928	60	2	13		12	1
1929	61		18	1	11	
1930	69	1	12		12	1
Total	403	7	128	3	71	2

Under the term "cyclomastopathy" fibro-adenomas and related lesions are considered by Oliver and Major, who found only 1 case of carcinoma developing among 106 cases followed over 5 years.

Campbell followed a group of 190 cases of simple cystic disease treated by local excision, 62 per cent for 5 years or more, and found 1 carcinoma of the breast as well as 1 case in which the patient was dying from carcinoma of unknown origin. Of 42 cases of adenocystic disease, 52 per cent were followed 5 years or more and no cases of carcinoma were found. Simple mastectomy was done in 57 patients, of whom 64 per cent were followed 5 years or more. One developed carcinoma in the opposite breast.

TABLE IV.—AGE OF TORONTO CASES AS OBTAINED

Diagnosis	Youngest—age in years	Oldest—age in years	Cases	Average age in years
Chronic cystic mastitis	21	64	188	40.9
Chronic mastitis	19	65	75	41.0
Duct papilloma	21	65	43	40.7

TABLE V. — MASSACHUSETTS 1930 FEMALE BREAST CANCER AGE SPECIFIC DEATH RATES BY MARITAL STATUS*

Age in years	Single		Not single		Total	
	Deaths / Population	Rate per 100,000	Deaths / Population	Rate per 100,000	Deaths / Population	Rate per 100,000
Under 30	3 / 919,448	0.3	2 / 176,558	1.1	5 / 1,096,006	0.5
30–39	7 / 68,225	10.3	37 / 271,258	13.6	44 / 339,483	13.0
40–49	30 / 46,689	63.4	83 / 236,556	35.0	113 / 283,245	40.0
50–59	40 / 36,956	108.1	121 / 184,928	65.5	161 / 221,884	72.6
60–69	44 / 23,816	190.0	125 / 122,548	102.0	169 / 146,364	115.5
70 and over	27 / 13,793	196.0	127 / 75,748	168.0	154 / 89,541	173.8
Total over 30	148 / 189,481	78.1±6.4	494 / 891,036	55.5±2.5	642 / 1,080,517	59.3
Total over 40	141 / 121,256	116.1	457 / 619,778	73.8	598 / 741,034	80.5
Total all ages	151 / 1,108,929	13.6†	497 / 1,067,594	46.5	648 / 2,176,523	29.7

*Based on three year average of female breast cancer deaths centering on 1930—1930 U. S. Census Population by age and marital status used.
†This low rate due to large number of female children.

Among 54 patients on whom partial mastectomy was performed for chronic cystic mastitis and followed from 2 to 11 years, Klingenstein found 2 carcinomas of the breast. Eleven of the patients had multiple operations for chronic cystic disease.

The relation of ovarian hormones to chronic cystic mastitis has been considered by many, among them Lewis and Geschickter (24), and Taylor, who believes that an ovary producing estrin must be present in any case with painful, nodular breasts. He also stresses the importance of local vascular congestion in the development of the lesion.

There has been a growing tendency to regard these lesions as not necessarily leading to malignancy. Bloodgood (6) has had much influence in advancing this viewpoint. In 1921 he reported subsequent carcinoma developing in only 3 of 128 cases of chronic cystic mastitis treated by local excision. Later in a series

TABLE VI.—DURATION OF FOLLOW-UP IN 604 CASES

Age in years	Average duration of follow-up in years
—29	6.9
30 to 39	8.4
40 to 49	8.6
50 to 59	10.1
60 to 69	11.2
70 to 79	9.4
80 and over	10.9
Unknown	10.5
Total under 50	8.4
Total over 50	9.9

TABLE VII.—AGE DISTRIBUTION OF CASES OF BREAST DISEASE FOLLOWED

(Age at end of follow-up period)

Age in years	Number of cases
—29	13
30 to 39	61
40 to 49	155
50 to 59	217
60 to 69	87
70 to 79	35
80+	10
Unknown	26

of 500 single, blue-domed cysts he found (7) only 5 carcinomas.

A recent article by Lewis and Geschickter (25) further emphasizes this view. Three deaths from breast cancer occurred among 271 cases of "adenosis" followed more than 5 years, and only 1 death from breast cancer among 252 cases of cystic disease followed more than 5 years. In 54 cases in the latter group biopsy had not been done. Their figures will be discussed later in the paper. Based on a study of 2,675 cancers of the breast, they state that only 0.5 per cent of the usual type show in the surrounding breast tissue changes characteristic of Schimmelbusch's disease, yet 30 per cent of the comedo type of cancer are associated with breast tissue showing these changes. They conclude: "These figures would seem to indicate that cystic disease and adenosis are not precancerous lesions and that radical operations should not be performed as frequently as they are. Another form of therapy should be tried."

In this connection, Rogers and Nathanson noted that in half their cases of chronic cystic mastitis treated with endocrine therapy (ovarian residue and/or progynon) palpable lumps disappeared.

MATERIAL

To carry out this analysis a study was undertaken of women whose breasts had been operated upon in various Boston hospitals. These were supplemented by a study in Toronto by Dr. J. R. E. Morgan of a group of 602 women whose breasts had been operated upon in the Toronto General Hospital,[1] 419 of the group being followed for 5 years or more.

The results obtained by Dr. Morgan are presented in Tables III and IV.

The 602 cases are arranged by years and diagnoses in Table III, to illustrate the types of lesions encountered and the decreasing tendency to call certain changes "precancerous." Due to duplication of lesions in a single breast, the total of the tabulated lesions—614—does not check with the number of cases—602.

The frequency with which carcinoma developed subsequently in these breasts is shown in Table III. Ten cases of cancer were found, an incidence of 1.7 per cent. In 337 cases a portion of breast tissue less than 3 centimeters in diameter was removed; in the remainder complete unilateral mastectomy was done. In 23 additional cases diagnosed as "precancerous," two breast cancers developed. Of the total 12 cases of carcinoma, 10 are dead and 2 living, 6 and 8 years after operation, respectively.

The ages of these patients could not be obtained with accuracy, and so this group has not been used for detailed study. The average age of the patient is 41 years (see Table IV), which may account in part for the somewhat lower incidence of cancer than was found in the two Boston samples, which had an average age of 52 years.

The Massachusetts group was at first divided in two parts to offset such possible confusing points as variation in popularity of clinics or in types of patients attending different clinics. One group consisted of patients from Boston City, Peter Bent Brigham, and Huntington Hospitals; the other group came from New England Deaconess and Pondville Hospitals and those hospitals using the state tumor diagnosis service. Since no significant

[1] We are indebted to the late Dr. Oskar Klotz for permission to use this material.

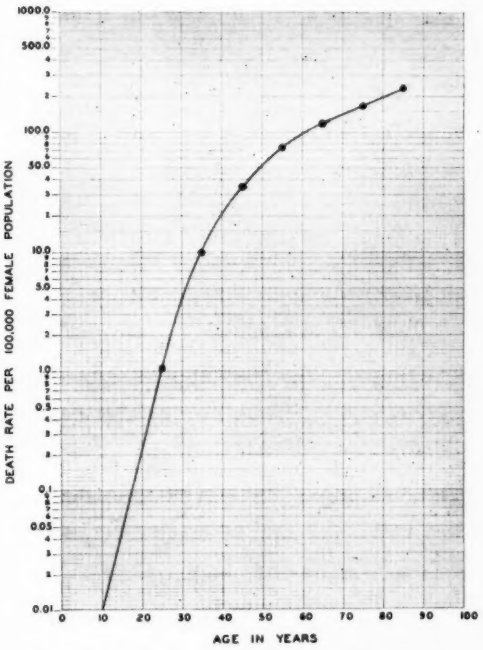

Fig. 1. Graph illustrating increase in breast cancer death rate with age. Semi-logarithmic scale.

difference appeared in the two samples, they are lumped together in most calculations.

While over 2,500 various non-malignant lesions of the female breast were reviewed in studying the Massachusetts material,[2] follow-up was attempted in about 900, as indefiniteness of identification and length of time elapsed made many cases unsuitable. The periods after operation on the breast in the 604 cases successfully followed ranged from 5 to 21 years. The follow-up periods with most cases are the sixth and seventh years—164—and from the eleventh year on—135.

These cases have been divided, perhaps arbitrarily, into groups of chronic mastitis, 173 cases; chronic cystic mastitis, 340 cases; adenocystoma, 21 cases; and adenofibroma, 70 cases.

The chronic mastitis group is comparable to the fibro-adenomatosis simplex of Semb,

[2] I am indebted to Drs. H. M. Clute, E. M. Daland, J. Fallon, F. H. Lahey, C. C. Lund, L. S. McKittrick, F. Parker, Jr., G. W. Taylor, I. J. Walker, S. B. Wolbach, and many others for assistance, pathological material, and clinical information; to Drs. Herbert L. Lombard and E. B. Wilson for statistical advice and aid.

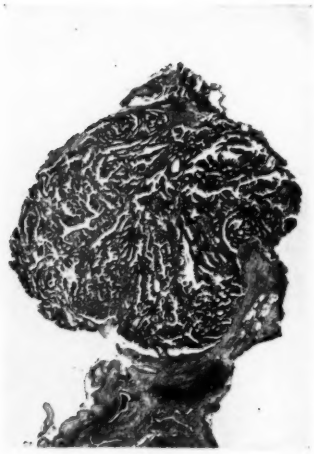

Fig. 2.

Fig. 3.

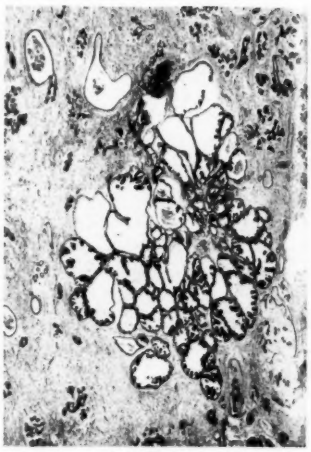

Fig. 4.

Fig. 2. Adenocystoma of breast. This mass shelled out of a duct beneath the nipple. Simple mastectomy (left), female, 67 years of age, No. 37428. ×6.5.

Fig. 3. Adenocystoma of breast. Note active but orderly epithelium. Associated with marked chronic cystic mastitis. Local excision, female, 58 years of age, No. 39155. ×120.

Fig. 4. Chronic cystic mastitis: multiple small cysts with slight epithelial hyperplasia. Large cysts also present. Local excision, female 45 years of age, No. 37813. ×7.5.

the breast with diffuse or local thickening due to changes of both epithelium and connective tissue. Cysts of microscopic size may be present. Some of the more marked cases of mazoplasia, as defined by Cheatle and Cutler, would fall into this group.

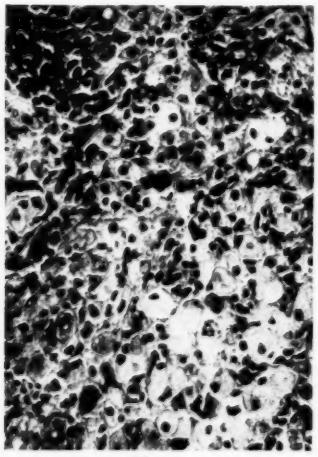

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 5. Chronic cystic mastitis: lipoid-filled phagocytes. Local excision, female, 40 years of age, No. 38958. ×235.

Fig. 6. Chronic cystic mastitis. Focus of small cysts with slight epithelial hyperplasia. Local excision. Patient subsequently developed carcinoma of this breast for which radical mastectomy was done. Also had malignant adenoma of descending colon. Female, 68 years of age, No. 36178. ×80.

Fig. 7. Chronic cystic mastitis showing epithelial hyperplasia. Six weeks previously intraductal papillary adenocystoma removed from same breast. Simple mastectomy. Female, 66 years of age, No. 37758. ×235.

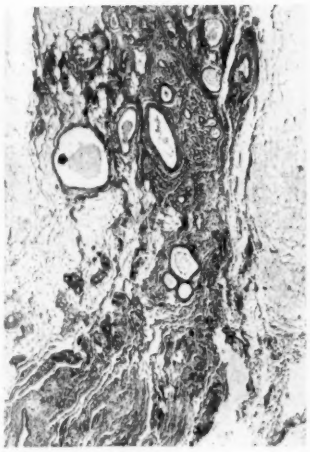

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 8. Very slight cystic disease of the microcystic type. Local excision, female, 61 years of age, No. 44184. ×19.

Fig. 9. Chronic cystic mastitis with diffuse acinar hyperplasia. Local excision, female, 23 years of age, No. 26–1264. ×19.

Fig. 10. Marked epithelial hyperplasia, precancerous. Local excision, female, 20 years of age, No. 38876. ×120.

Chronic cystic mastitis includes lesions recognized by such descriptive terms as Schimmelbusch's disease, fibro-adenomatosis cystica of Semb, the cystipherous desquamative epithelial hyperplasia of Cheatle and Cutler, Aschoff's mastopathia cystica, and the maladie kystique of Reclus. These breasts are nodular due to the presence of one or more cysts with varying degrees of epithelial hyperplasia and papillary ingrowths. Clear columnar acidophilic cells may be conspicuous, often with fibrosis and thickening of periductal elastica.

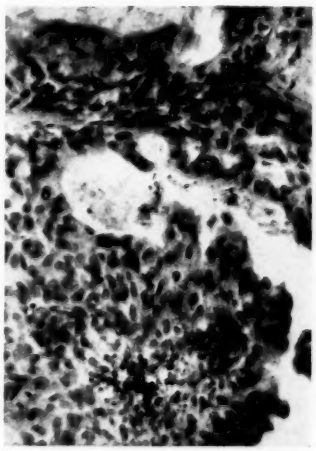

Fig. 11. Carcinoma developing in chronic cystic mastitis. Radical mastectomy, female, 66 years of age, No. 28–790. ×120.

Fig. 12. Intracanalicular adenofibroma of the breast. Local excision was performed, female, 32 years of age, No. 37806. ×19

The term adenocystoma is confined to true intraductal papillary growths, commonly in the ampulla or larger ducts. It does not include the papillary projections of chronic cystic mastitis.

Adenofibroma, or adenoma, is the pseudoencapsulated tumor so common in adolescence made up of varying proportions of stromal and epithelial elements. They may or may not be intracanalicular in arrangement.

These last two groups are included to provide a contrast to the groups of chronic mastitis and chronic cystic mastitis and will not be considered in all the tables.

Of the lesions present, 347 had been treated by incomplete mastectomy, ranging from portions a few millimeters in diameter to 3 centimeters in most; even to almost complete mastectomy in rare instances. Complete local mastectomy was done in 163 cases, and conclusions are based in these on the behavior of the remaining breast.

In this study the mode of research was based on three assumptions: first, that removal of a lump in the breast which proved on excision to be chronic mastitis or chronic cystic mastitis did not necessarily remove all the abnormal tissue; second, that alterations in the breast are frequently produced by hormonal influences which may be fairly assumed to continue to affect at least some other portions of the breast tissue after one portion has been removed; and finally, that if chronic mastitis, cystic or otherwise, is a precursor of carcinoma, those women who had this disease, proved by partial or unilateral mastectomies, should have a higher frequency of carcinoma of the breast than women in the general population.

A weak point lies in that supposedly the most abnormal breast tissue has been removed by the biopsy, and thus an artificially low incidence of subsequently developing carcinoma may be obtained. It may also be objected that actual carcinomas may have been removed in the course of the biopsy or mastectomy, and any subsequently developing cancer might be a mere recurrence. This objection can be largely answered by the gross and microscopic studies of the tissue removed which failed to show carcinoma.

The most difficult problem is to establish a fair norm for the morbidity rate of female breast cancer. Fortunately there has been active interest in the incidence of breast cancer in Massachusetts for years, and we believe that the figures for this state are as accurate as now can be obtained.

Although no satisfactory figures exist for breast cancer morbidity rates, we may assume that the annual mortality rate reflects the annual attack rate minus those cases in which patients were cured and those in which diagnosis was never made; we have used this as a basis for our calculations. In order to allow for those patients cured, about 20 per cent of all cases seen, and those never diagnosed—unknown, but probably not large in the breast cancer group, owing to readily available diagnostic facilities and widespread educational efforts in Massachusetts—we have estimated the annual attack rate at twice the death rate.[1]

The age specific death rates for mammary cancer in Massachusetts women in 1930 are shown in Table V. In keeping with other figures (22), the rate is significantly greater for single women, 78.1 ± 6.4 per 100,000 for those single over 30 years of age as against 55.5 ± 2.5 per 100,000 for those not single. The rate for all women over 30 years of age is 59.3 per 100,000. Doubling the death rate for all females (29.7 per 100,000) and for those over 30 years of age (59.3 per 100,000) gives an annual attack or morbidity rate of 0.06 per 100 for the total Massachusetts 1930 female population, and a rate of 0.12 per 100 for those women over 30 years of age. While this may be too high, it is well from the standpoint of this study to overestimate rather than underestimate the number of breast cancers occurring in the total female population, as our preliminary data indicated that more breast cancers than usual develop in the chronic mastitis group.

Since some of the women in the group of chronic mastitis—50—and in the group of chronic cystic mastitis—156—had had one breast amputated, the expected rate should perhaps be halved for these, as they had only

[1] Actually 1.5 times the death rate is probably more accurate, but every effort is made to keep the calculations conservative.

TABLE VIII.—OCCURRENCE OF CANCER BY AGE AND PRE-EXISTING LESION

	Under 30 yrs.		30–39 yrs.		40–49 yrs.		50–59 yrs.		60–69 yrs.		70–79 yrs.		80 yrs.+		Unknown		Total	
	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years
Chronic mastitis—incomplete	6	40	18	178	35	320	42	429	12	162	7	85	1	5	2	23	123	1242
Chronic mastitis—complete	0	0	4	33	7	62	18	185	15	160	2	10	2	17	2	25	50	492
Chronic cystic mastitis—incomplete	3	19	20	134	56	441	77	670	19	210	5	37	2	26	2	11	184	1548
Chronic cystic mastitis—complete	1	6	8	60	33	243	64	551	33	348	13	108	4	56	0	0	156	1372
Adenoma	0	0	0	0	9	100	2	25	3	37	5	50	1	5	1	11	21	228
Adenocystoma	3	25	11	107	15	161	14	160	5	70	3	40	0	0	19	202	70	765
Total of all cases	13	90	61	512	155	1327	217	2020	87	987	35	330	10	109	26	272	604	5647
Number of cancers		0		2		9		8		4		4		2		1		30

half the normal amount of breast tissue in which tumor could develop.

That this last assumption is not entirely fair is shown by the occasional finding subsequently in the operative field of normal or pathological breast tissue which may even develop carcinoma after a breast amputation. Consequently the basis of calculation has been left unchanged.

The Massachusetts cases of breast disease studied were divided into two samples, to see whether the trends were similar in each group, and to offset such possible confusing points as variation in popularity of clinics or in types of patients attending different clinics. No significant difference appeared in the two samples, and so they are lumped together in most calculations.

The Massachusetts material when considered in aggregate (sample I and sample II combined) contains 604 cases, in 30, or 2.6 per cent, of which breast cancer developed. The average follow-up period was 9.3 years. There were 173 cases of chronic mastitis with 11 carcinomas, 340 cases of chronic cystic mastitis with 14 carcinomas, 21 cystadenomas with 3 carcinomas, and 70 adenomas with 2 carcinomas. These last two groups are too small to be statistically significant, but are included for the sake of completeness.

The length of follow-up is of great importance. Of the 604 cases, 13 were followed an average of 2.7 years and all these were patients who developed cancer. The other 17 cancer cases were followed an average period of 9.3 years. This is exactly the same as the average period for all cases of breast disease. There is no appreciable variation in length of follow-up with age (Table VI). The longest follow-up period for any case is 21 years. Three per cent, or 17 cases, were followed 19 years and over, and 12 per cent, or 72 cases, were followed 15 years or over.

The age distribution of the cases is important, as disproportionately few cases in the older age groups would materially lower the expected number of cases of cancer. Table VII gives the age distribution of the Boston cases and shows the sample has an adequate number of cases in the later decades (349 over 50 years of age) to have a fair proportion in the so called "cancer age."

The great importance of age of the cases studied is strikingly brought out by Figure 1,

TABLE IX.—INCIDENCE OF BREAST CANCER IN CHRONIC MASTITIS AND CHRONIC CYSTIC MASTITIS

(Boston Samples I and II plus Toronto Sample)

	Number of cancers	Number of individuals	Average duration of follow-up years	Number of risk years	Rates (per 100)	
					With risk years adjustment	Without risk years adjustment
Chronic mastitis	14	301	10.0	3014	0.47±0.12	4.65±1.23
Chronic cystic mastitis	21	743	8.6	6379	0.33±0.07	2.83±0.61
Total	35	1044	9.0	9393	0.37±0.06	3.35±0.56

TABLE X.—COMPARISON BETWEEN AGE SPECIFIC CANCER ATTACK RATES FOR WOMEN WITH BREAST DISEASE AND NORMAL WOMEN
(Rate per 1,000 risk years)

	Under 30 yrs.		30–39 yrs.		40–49 yrs.		50–59 yrs.		60–69 yrs.		70–79 yrs.		80 yrs.+		Unknown		Total	
	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years	No. persons	No. risk years
Total of all cases	13	90	61	512	155	1327	217	2020	87	987	35	330	10	109	26	272	604	5647
Number of cancers	0		2		9		8		4		4		2		1		30	
A. Cancer attack rate per 1,000 risk years	0		3.91		6.79±2.26		3.96		4.05		12.10		18.35		3.68		5.32±0.97	
B. Cancer attack rate per 1,000 Massachusetts female population	0.01		0.26		0.80±0.05		1.45		2.31		3.12		4.51		0		1.19±0.03	
Difference between rates (A—B)			3.65		5.99±2.26		2.51		1.74		8.98		13.84				4.13±0.97	
Number of times A. rate is greater than B. rate			15.0		8.5		2.7		1.8		3.9		4.1				4.5	

	(1) 30–49 years	(2) Over 50 years	Number of times (2) is greater than (1)
A. Cancer attack rate per 1,000 risk years	5.96±1.80	5.23±1.23	0.88
B. Cancer attack rate per 1,000 Massachusetts female population	0.51±0.03	2.11±0.07	4.14
Difference between rates (A—B)	5.45±1.80	3.12±1.23	
Number of times A. rate is greater than B. rate	11.7	2.5	

which shows the age specific breast cancer death rates for Massachusetts women averaged for the years 1928 to 1932. This is presented on semi-logarithmic paper, in order to keep the scale compact.

Unfortunately we have no way of knowing how many of the women who have developed chronic mastitis and related lesions come under observation. We may assume, however, that a large proportion seek medical aid.

That by no means all cases of breast cancer develop following chronic mastitis is obvious to anyone treating an appreciable number of such lesions. Since the cases of breast cancer occurring in a given population represent cancer arising in previously non-diseased breasts plus those occurring in breasts the site of previous disease, any significant increase in the breast cancer rate in the latter group over that of the general population would imply such a group to be definitely predisposed to development of breast cancer.

In the subsequent tables will be presented the evidence for considering chronic mastitis to be a condition predisposing to the development of breast cancer.

Table VIII gives by age and pre-existing lesion the number of cancers developing in the 604 Massachusetts cases. Both the number of women in each age group and the number of risk years (number of women multiplied by average follow-up period in years) are given. The cases of chronic mastitis and chronic cystic mastitis are further divided into those having had a portion of breast tissue removed and those having had a more or less complete unilateral mastectomy.

TABLE XI.—SAMPLES I AND II (COMBINED)
(Summary sheet of Boston Cases)

	Chronic mastitis	Chronic cystic mastitis	Adeno-cystoma	Adenoma	Total
Number of women	173	340	21	70	604
Number of risk years	1734	2920	228	765	5647
Average duration (in years)	10.0	8.6	10.8	10.9	9.3
Cancer rate per 1000 risk years	6.4	4.8	13.2	2.6	5.3

TABLE XII.—COMPARISON OF OBSERVED NUMBER OF CANCERS OF BREAST IN WOMEN WITH BREAST DISEASE AND EXPECTED NUMBER OF SUCH CANCERS IN NORMAL WOMEN

	Observed	Age adjusted expected		Broad age adjusted expected*		Crude expected†	
		Adjusted for follow-up period	Without follow-up adjustment	Adjusted for follow-up period	Without follow-up adjustment	Adjusted for follow-up period	Without follow-up adjustment
Chronic mastitis Samples I and II, Incomplete	7	1.6	0.15	1.47	0.15	0.74	0.07
Complete	4	0.8	0.08	0.58	0.06	0.29	0.03
Total	11	2.4	0.23	2.06	0.21	1.03	0.10
Canada total	3			1.52	0.15	0.76	0.08
All groups total	14			3.58	0.36	1.79	0.18
Chronic cystic mastitis Samples I and II, Incomplete	7	1.8	0.23	1.83	0.22	0.92	0.11
Complete	7	2.4	0.26	1.63	0.18	0.82	0.09
Total	14	4.2	0.49	3.46	0.40	1.74	0.20
Canada total	7			4.10	0.48	2.06	0.24
All groups total	21			7.56	0.88	3.80	0.44
Chronic mastitis and chronic cystic mastitis Samples I and II total	25	6.6	0.72	5.52	0.55	2.50	0.28
All groups total	35			11.14	1.24	5.59	0.69
Adenocystoma Sample I total	3	0.4	0.04	0.27	0.025	0.14	0.01
Canada total	2			0.91	0.08	0.46	0.04
Adenoma Sample I total	2			0.91	0.08	0.45	0.04
All cases Samples I and II total	30			6.70	0.72	3.36	0.36
Canada total	12			6.54	0.71	3.27	0.36
All groups total	42			13.16	1.43	6.63	0.72

*Broad Age Adjustments based on rate for Massachusetts females over 30 years of age (.0011865).
†Crude Expected based on rate for total Massachusetts females regardless of age (.0005945).

In this group of 604 cases which might well be assumed to show a lower incidence of breast cancer than the general female population, since that portion of breast tissue noted to be abnormal had been removed, there were 30 cases of cancer developing in a total of 5,647 risk years. It may be objected that the striking number of cancers occurring in this group is due to the inclusion of the 21 cases of adenoma and the 70 cases of adenocystoma. However, only 5 cancers were associated with these—3 with the adenocystomas and 2 with the adenomas.

Among all the chronic mastitis and chronic cystic mastitis cases included in this study, lumping together both the Boston figures and the Toronto figures, one finds 35 cancers occurring in 1,044 individuals, representing a total exposure of 9,393 risk years (Table IX). This gives the startlingly high 3.35 per 100 as a crude rate, and 0.37 per 100 as a corrected or annual attack rate, taking the follow-up period into consideration. This contrasts with an average[1] mortality rate of 0.029 per 100 which is based on the Massachusetts female population in 1930, or morbidity rate of 0.058 per 100.

When the age specific cancer attack rates per 1000 risk years of the chronic mastitis group are calculated and contrasted (Table X) with those per 1000 risk years of Massachusetts female population two facts stand out. The cancer rate for women with pre-existing breast lesions is 4.5 times as great as for all women; and this predominance is especially marked in the decades below 50 years of age.

[1] Average for 3 years centering on 1930.

TABLE XIII.—COMPARISON OF FEMALE BREAST CANCER RATES, EXPECTED AND OBSERVED PER 100

	Massachusetts female crude attack rate	Massachusetts age adjusted attack rate (females over 30)	Observed attack rate with follow-up adjustment	Observed attack rate without follow-up adjustment
Chronic mastitis Samples I and II, Incomplete	0.06	0.12	0.56±0.21	5.69±2.09
Complete	0.06	0.12	0.62±0.41	8.00±3.83
Total	0.06	0.12	0.64±0.19	6.35±1.86
Canada total	0.06	0.12	0.23±0.14	2.34±1.34
All groups total	0.06	0.12	0.47±0.12	4.65±1.23
Chronic cystic mastitis Samples I and II, Incomplete	0.06	0.12	0.45±0.17	3.81±1.41
Complete	0.06	0.12	0.51±0.19	4.49±1.66
Total	0.06	0.12	0.48±0.13	4.12±1.07
Canada total	0.06	0.12	0.20±0.08	1.74±0.65
All groups total	0.06	0.12	0.33±0.07	2.83±0.61
Chronic mastitis and chronic cystic mastitis Samples I and II, total	0.06	0.12	0.54±0.11	4.87±0.95
All groups total	0.06	0.12	0.37±0.06	3.35±0.56
Adenocystoma Sample I total	0.06	0.12	1.32±0.76	14.3±7.65
Canada total	0.06	0.12	0.26±0.18	2.82±1.97
Adenoma Sample I total	0.06	0.12	0.26±0.19	2.86±2.00
All cases Samples I and II total	0.06	0.12	0.53±0.10	4.96±0.88
Canada total	0.06	0.12	0.22±0.06	1.99±0.56
All groups total	0.06	0.12	0.38±0.06	3.48±0.53

Thus, in the age group 30 to 49 years, the cancer attack rate per 1000 risk years in the group being studied was 5.96±1.80 as contrasted with a calculated attack rate for the Massachusetts female population of 0.50±0.03, a rate of 11.7 times as great for the women with pre-existing breast lesions as for the Massachusetts female population in the same age group. This preponderance is less striking in the older age groups. In cases over 50 the rate in the study group is slightly less, 5.23±1.23, as contrasted with a considerably greater rate for the Massachusetts female population, 2.11±0.07, still giving a preponderance of 2.5 times as high a rate in the group with pre-existing breast lesions as compared with women in general.

Thus, not only is cancer more prevalent in those women who have given evidence of previous breast pathology, but in addition this preponderance is particularly marked in the women under 50 years of age, at the time when the breast tissue is more definitely responsive to endocrine influences than in the later years of life. In other words, *once a woman who has developed chronic mastitis reaches the menopause, her chance of developing breast cancer is not much greater than that of anyone, but until that time, she is in far greater danger of developing breast cancer than is the woman whose breasts have been apparently normal.*

If we still further subdivide the difference in rate by age groups, as is also given in Table X, we note that there is a general trend for the discrepancy between the two sets of rates to be higher in the younger age groups and to decrease in the older. However, in every age group the rate is higher for the patients with pre-existing breast pathology than the rate for the entire female population, and when all the cases are lumped regardless of age there is 4.5 times as high a rate among the group of cases studied as among the Massachusetts female population.

In order to determine which lesion, if any, presented the greater risk, Table XI was prepared, in which the cases studied are classified by lesions and the rate per 1,000 risk years calculated for each. The rates differ considerably from one another, except in the two types of mastitis, and give some support for the belief held by many pathologists that the adenocystomas are more dangerous than the adenomas, patients in whom the former had occurred having a rate of 13.2 per 1,000, the latter a rate of 2.6 per 1,000.

Still another way of comparing the frequency of the cancer cases in the group studied, as contrasted with the total Massachusetts female population, is a direct comparison of the observed number of cases as contrasted with the expected number. This is presented in Table XII. These are subdivided according to the types of lesions, the sample of the population, and the type of treatment. The observed number of cancers in any given group is contrasted with the expected number of cancers presented in three different ways. First, a specific age adjustment; second, a broad age adjustment for females over 30 years of age; and third, a crude rate without any adjustment for age, based on the total Massachusetts female population. The expected number of cancers were calculated by the following formula: The mortality rate was multiplied by two to give an attack rate, as explained earlier. The rate was then dropped back 2 years as a further correction, since the observed cases of cancer were based on the time of appearance of the cancer and not on the death of the patient, on the assumption that an average duration of untreated breast cancer is 2 years from the time the lesion is diagnosed to death.[1]

It will be noticed that in every age group and in every lesion and in every sample, the observed number of cases materially exceeded the expected. Forty-two cancers were observed as against 13.16 expected in the grand total of all cases. In the Boston samples of chronic cystic mastitis and chronic mastitis 25 cases of cancer occurred, as against 6.6 expected. Thus this method of calculation substantially agrees with the other in giving a fourfold increased prevalence of breast cancer in those women with antecedent chronic mastitis.

Another way of contrasting the incidence of carcinoma in the total female population and in the chronic mastitis group is presented in Table XIII. The expected cancer attack rate per 100 and the actual attack rate are compared. Here the various population samples and the various types of breast lesions have been considered separately and the cancer attack rate determined for each, then contrasted with both a crude expected attack rate for the total Massachusetts female population and a rate calculated for Massachusetts females over 30 years of age. The crude rate is 0.06 per 100 for the total female population and the adjusted for those of age over 30 is 0.12 per 100. The observed attack rates are calculated with adjustment for follow-up years and without adjustment for follow-up years. Utilizing this method of calculation as well, it is seen that in every sample the observed attack rate is greater than the calculated attack rate, although 100 is 4.5 times as great as the expected attack rate adjusted for the Massachusetts female population of above 30 years of age. This observed rate is significantly greater than the expected.

EVALUATION OF STUDY

The foregoing data indicate that women with breast abnormalities of the types considered, particularly chronic mastitis, chronic cystic mastitis, and papillary adenocystoma, show a definite predisposition to the development of breast cancer, especially prior to the menopause. This is despite the fact that the most abnormal or even all the abnormal tissue had been supposedly removed by surgical intervention.

Statistical studies are handicapped from the outset by an ignorance of the course of the benign lesions of the breast, their cause and incidence in the general female population. But unless a very large proportion of clinically significant mammary pathology is overlooked, the conclusions from this study would not be vitiated. In a careful search of breast tissue at autopsy, it is possible to find small foci of

[1] This should be distinguished from the figure of about 3 years for average duration of symptoms to death.

chronic mastitis in a surprisingly high proportion of cases, far higher than could be detected by any method of clinical examination. Such foci might be a starting point for carcinoma, but there is no way to prove or disprove the point. It is to be hoped that a careful study may be made that will give definite information as to the incidence of clinically significant chronic mastitis and chronic cystic mastitis in the general population.

While the findings here presented corroborate those of many earlier workers in establishing a relationship between "chronic mastitis" and carcinoma (16, 17, 31, 34, 38), they conflict with some recent work, notably the conclusions of Lewis and Geschickter (25). These authors present the incidence of cancer of the breast among 1,048 cases of "adenosis" and cystic disease, of which 523 patients were followed 5 years or more. Of the 1,048, 381 patients were not operated on, but were diagnosed clinically. It was not stated how many of the patients checked by pathological examination were followed. The ages of their patients are not given in the text, but based on the graph they present, the average age of their cases is 38 years, an age at which the development of breast cancer is relatively rare. It is not stated whether the graph represents ages at the beginning or end of the follow-up period. The rate of breast cancer they find for the followed cases I calculate as 0.77 ± 0.4 per 100 for the total period, or about 0.15 per 100 per year. If the ages of their patients represent the end of the follow-up period, the annual death rate for the total Massachusetts female population of the same age is 0.01 per 100 and doubled for a morbidity rate is 0.02 per 100. Thus the incidence of breast cancer in their cases is 7 times the expected. However, if the ages of their patients represent the beginning of the follow-up period, a mortality rate for all Massachusetts women of 0.04 per 100 or a morbidity rate of 0.08 would apply. In this instance, the incidence of breast cancer among their cases is twice the expected. On either basis of calculation their conclusion that "these figures would seem to indicate that cystic disease and adenosis are not precancerous lesions" appears unsupported.

In any evaluation of statistical material dealing with this subject, it is important to remember the finding of Bigelow and Lombard that the average age of females with breast cancer is 60 years. Therefore, any series of cases in younger women should not be compared with crude breast cancer rates, but with age-specific rates.

If we consider Figure 1, the strikingly increased incidence of cancer of the breast with increasing age becomes apparent. The women in older age groups must have less and less hormonal stimulation of mammary tissue. Therefore, it may well be argued that if chronic mastitis and chronic cystic mastitis, which seem to be definitely associated with hormonal disturbances, are important factors in the development of cancer, there should be a decrease in the age-specific cancer rate toward the later years of life rather than the increase that is definitely present. That the changes which the breast tissue may undergo in old age are of enormous importance in the etiology of breast cancer, no careful observer can deny. That the output of estrogen alone of the female sex hormones continues after the menopause may well be significant.

However, the genesis of cancer of the breast is not my immediate problem. I am concerned with the significance of abnormality of the breast tissue as evidenced in chronic mastitis and chronic cystic mastitis in relation to the subsequent development of cancer. It is immaterial in the present discussion that cancer of the breast is much more prevalent at 80 years of age than it is at 40. The point of definite importance is that 4.5 times as many cases of chronic mastitis subsequently developed cancer of the breast, age for age, as would be expected for the female population as a whole.

A relationship between chronic mastitis and cancer exists, regardless of the interpretations placed on it. Although cancer of the breast may often arise independent of the lesion, its significance as a precancerous process remains. Merely because a very high proportion of elderly people develop skin cancer, we cannot discount the effect of excessive exposure to radiation as a cause of skin cancer. In the rather rough classification possible in this study we have found the actively proliferating lesions such as intraductal papilloma and cysts

with papillary epithelium to be followed by cancer more often than the adenofibromas or large cysts with atrophic lining epithelium.

Cystic disease of the breast may lead to grave developments not only in humans but in laboratory animals, as has been so clearly pointed out by Greene in the rabbit, and as the experiments of Bagg in inducing epithelial stimulation and retention of secretion of the mouse breast tissue suggest.

One somewhat discouraging finding is that in general no one type of lesion in the human breast can be pointed out as particularly likely or unlikely to be followed by cancer, except the intraductal papilloma on the one hand and adenofibroma on the other. In general, the larger cysts with atrophy of their lining epithelium are less likely to be followed by malignancy than those with active, often papillary, epithelium. In such a cyst, aspiration of the contents may be a justifiable therapeutic procedure, though determination of the exact nature is not easy, and excision is preferable.

The difficulty of accurate diagnosis is well shown by a recent case, that of a woman 32 years of age, who had a small focus of thickening in her breast. This was thought by two competent surgeons to be benign, but on operation an adenocarcinoma, less than 1 centimeter in diameter, with metastasis to 1 axillary lymph node was found.

From the standpoint of this study, what shall we do about a woman who had developed a sufficiently marked degree of chronic mastitis, cystic or otherwise, to be of clinical importance? Hormonal therapy is often effective in the milder cases, and should be tried if the lesion is characteristic of chronic mastitis, cystic or otherwise, and does not suggest carcinoma. It must be remembered, however, that estrogenic hormones stimulate mammary epithelium. If prompt response to hormonal therapy is not obtained, the breast should be operated upon and the nature of the lesion determined. If mastitis is present, in the light of the data here presented, it is impossible not to regard the patient as being subject to special risk of the development of cancer of the breast at some later date, should her mammary tissues be allowed to remain. There are two courses open: One, the more radical (and the less desirable), is to advise the patient to permit amputation of both breasts, since, when the process has involved one, it may well involve the other. The other alternative is, after removal of the definitely diseased tissue, to have her breasts carefully checked at least every 6 months for evidence of abnormality. This, when encountered, should lead to a simple unilateral mastectomy if the lesion is benign, and radical mastectomy if proved malignant.

Even if not cancerous, breast tissue permitted to remain will probably develop further recurrence of the mastitic process. Not only is the hazard of carcinoma real, but the persistency of the mastitic process most striking. Not even in those cases in which temporization and incomplete surgical procedures have been carried almost to an extreme has there been any guarantee that with the piecemeal removal of pathological mammary tissue the residual tissue should remain free from disease. Thus, of 173 women with chronic mastitis in the Boston sample, 26 or 15 per cent had subsequent trouble with their breasts; and of 320 women with chronic cystic mastitis, 58 or 17 per cent had subsequent breast trouble. This corresponds with the 16 per cent recurrence rate noted by Greenough and Simmons (17). In other words, about 1 woman of 6, even after the most obviously diseased portion of her mammary gland is removed, develops subsequent trouble, ranging from serious discomfort to the requirement of a second operation for relief, and 1 of every 29 develops carcinoma.

If intraductal papilloma or adenocystoma (Figs. 2 and 3) is encountered, simple mastectomy should be seriously considered, although the figures in the present study are too few to permit conclusions to be drawn. Other lesions of similar type may be present in the rest of the breast, and the transition to cancer is relatively easily made. At times very careful pathological study is required to determine whether or not the epithelium is invading the surrounding tissue.

Not infrequently the typical picture of chronic cystic mastitis (Fig. 4) is complicated by various inflammatory changes. If, in such an event, much retained secretion is present,

foci of lipoid-filled phagocytic cells may constitute a prominent part of the picture (Fig. 5). As a rule, however, inflammatory changes play a minor rôle, and there is no evidence of transition from them to neoplasia. Epithelial hyperplasia (Figs. 6 and 7) is a much more important factor. At times the epithelium lining the cysts is in part or entirely acidophilic, usually high cuboidal or columnar (Fig. 6), and sometimes thrown up in papillary folds. This is similar to the epithelium of the so called sweat-gland carcinoma of the breast (23), which is better considered as originating from the mammary epithelium.

Sometimes the cystic disease is very variable, minimal in some portions (Fig. 8) and very well marked in others. The cysts may be associated with a diffuse proliferation of the acinar tissue, which gives a rather confusing picture (Fig. 9) that may simulate carcinoma.

Whether the cysts be large or small, their epithelium may be hyperplastic—at times extremely so, as in those in which only lack of invasion of the surrounding duct wall prevents a diagnosis of carcinoma (Fig. 10), but more often present to a minor degree. The papillary projections into the cysts may be uniradicular, but often show multiple points of origin. Different portions of the epithelial lining of the cysts may vary in cell size, shape, and staining reaction. Sometimes the epithelial proliferation forms broad projections, occluding the lumen save where it contains inspissated secretion. In such regions anaplasia may be marked, and definite carcinoma develop (Fig. 11).

Increase in periductal elastic tissue not infrequently occurs in chronic mastitis, but is not of value in determining whether or not carcinoma will develop subsequently. Breasts in which extreme degrees occur, as in intracanalicular adenofibroma (Fig. 12), develop breast cancer no more frequently than do all women.

SUMMARY AND CONCLUSIONS

1. The development of breast cancer in 1,206 women, whose breasts had been operated upon for benign non-infectious lesions of the breast, was studied and compared with the incidence of breast cancer in females of the general population in the same age groups. The figures for the Massachusetts female population for the 5 years centering on 1930 were used.

2. The results obtained from the 3 samples studied (2 in Boston and 1 in Toronto) were essentially similar.

3. The results obtained by different methods of statistical analysis are essentially similar.

4. While the data are insufficient with regard to adenoma and adenocystoma, they do indicate that adenoma is without significance as regards subsequent malignancy, and that adenocystoma is a precancerous lesion.

5. Estimates of the occurrence of breast cancer based on crude rates are of little value. Age specific rates are essential.

6. The breast cancer morbidity or attack rate may be estimated as double the mortality rate, based on Massachusetts figures.

7. The breast cancer attack rate is 0.12 per 100 for Massachusetts women over 30 years of age; but is 0.47 ± 0.12 per 100 for women with chronic mastitis; 0.33 ± 0.07 per 100 for women with chronic cystic mastitis; and 0.38 ± 0.06 for all cases studied.

8. When age specific rates are used, the breast cancer attack rate for women with chronic mastitis and related lesions in the age group from 30 to 49 years is 11.7 times the rate for the Massachusetts female population; in the group over 50 years of age, 2.5 times as great; in the entire group, 4.5 times as great.

9. Chronic cystic mastitis and chronic mastitis predispose to the development of breast cancer.

10. No means at present exist to determine those lesions which will develop cancer and which will not.

11. The risk is not sufficiently great to warrant bilateral mastectomy in cases of chronic mastitis and chronic cystic mastitis.

12. Patients in whom these lesions are found on operation should subsequently be carefully watched and simple mastectomy done if nodules or cysts develop in residual breast tissue.

13. The figures for incidence of cancer in the group studied although high are none the less minimal for the material and probably

with the further lapse of time additional cases will develop.

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SPLEEN EXTRACT IN THE TREATMENT OF TRANSPLANTED AND SPONTANEOUS MALIGNANT TUMORS IN MICE

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IN a recent paper (7) one of us (Lewisohn) presented the effect of subcutaneous injections of concentrated beef spleen extract (100 grams in 1 cubic centimeter) upon mouse sarcoma 180. It is well known that this tumor is most resistant to any form of treatment. The fact that a complete disappearance of the tumors resulted in 60 per cent of the animals demonstrated that concentrated spleen extract offered a new approach to a therapy of this very malignant tumor.

Additional observations reported in that paper were: (1) Whereas these tumors regressed in response to highly concentrated spleen extract their growth was stimulated by a weak concentration; (2) a marked increase in the size of the spleen was noted in animals treated with concentrated spleen extract, while neither animals treated with other organ extracts nor the controls showed an enlargement of the spleen. (3) The spleen extract did not elicit the Schwartzman phenomenon.

The fact that in general abdominal carcinomatosis the spleen is usually free from any metastasis has induced many research workers to study the effect of the spleen on malignant tumors. The literature on this subject is so extensive that it would be impossible to attempt a detailed report of previous publications. Suffice it to say that while retardation of growth in transplanted malignant tumors in animals has been frequently noted, no complete and lasting disappearance of these tumors had heretofore been reported.

Apolant showed over 25 years ago that the resistance of the body against tumor cell transplantation is lessened after splenectomy.

Urban and Schnitzler stated that intracutaneous transplantation of carcinoma or sarcoma is effective only after the reticulo-endothelial system has been blocked by a preliminary injection of a dye.

Brueda reported that he could grow mouse sarcoma or carcinoma in rats in 80 to 90 per cent of 200 animals after a splenectomy. However, he observed that this high percentage of takes could be obtained only during the first 2 weeks following splenectomy. Later other organs, for instance the liver, adopt the function of the spleen. This observation points toward the close relationship of the different organs constituting the reticulo-endothelial system.

Fischer-Wasels reported that he succeeded in causing small tumors to disappear after treatment with spleen extract. However, these tumors recurred as soon as the injections were interrupted. This is at variance with our observations that following the treatment with "concentrated" spleen extract 60 per cent of the tumors (sarcoma 180) not only disappeared, but did not recur after the treatment was stopped (observation time of many months).

Sugiura tested the action of aqueous extracts of different organs on mouse sarcoma 180. Spleen extract was found to exert the most destructive action upon the growth capacity of mouse sarcoma 180, while lung and placenta showed the least effect. Immersion of tumor fragments for 120 hours in spleen extract produced complete inhibition of growth.

The inhibitory action of spleen tissue upon tumor growth was recognized as early as 1912, when Oser and Pribram found that transplantable rat sarcoma grew more rapidly in splenectomized animals and that retrogression and cessation of tumor growth could be induced in sarcoma bearing rats by the subcutaneous injection of spleen-pulp.

Braunstein reported that he was under the impression that in cancer the spleen was the

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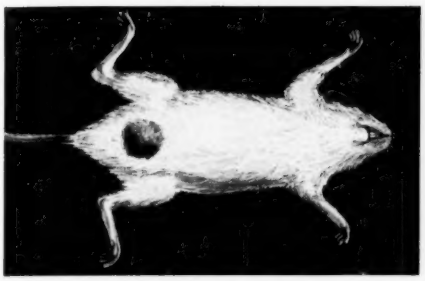

Fig. 1. Mouse 123. Treatment started February 18, 1939; healed May 29.

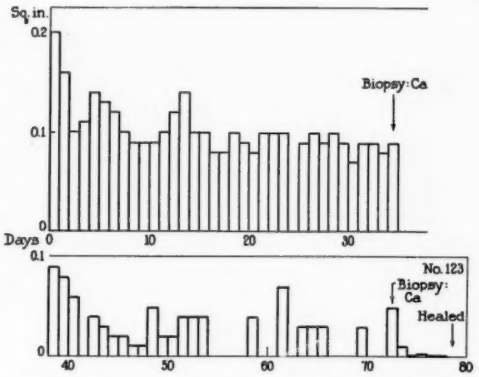

Fig. 2. Diagram of mouse 123. Injections of healed spleen extract—33 intravenous and 19 intraperitoneal—were given.

main seat for the formation of antibodies. He injected mouse carcinoma into normal mice and splenectomized these animals after 4 to 6 days. The spleens thus removed were used in the treatment of transplanted mouse carcinomas. The spleens were pulverized, dissolved in physiological sodium chloride solution, and injected three to four times daily into the carcinomatous animals. He was impressed by the fact that in animals thus treated the percentage of cures was higher than in the controls.

Biach and Weltmann showed that when a mixture of sarcoma and spleen was inoculated the growth of the tumor was inhibited and that the spleens of sarcoma-bearing rats suppressed the growth much more effectively than those from normal rats.

Mottram and Russ observed that when Jensen's sarcoma was mixed with spleens taken from rats whose tumors had undergone spontaneous regressions, the growth of the sarcoma was delayed.

A few years ago Woglom reported similar interesting experiments. He found that when he mixed fresh tumor emulsion of a rat sarcoma 39 with minced spleen from an immune rat, i.e. a rat in which the tumor had undergone a spontaneous regression, further growth of the tumor cells was markedly inhibited. The inhibition of the growth was proportional to the strength of the spleen emulsion. Thus the inhibition was greater when three parts of immune spleen to one part of tumor rather than equal parts of both constituents were used. These experiments pointed to the presence of a healing factor in the spleen of animals with sarcoma 39 which had undergone spontaneous regression.

In the previous paper (7) results in 281 animals were reported. Of these 170 were healed. These animals were kept for many months in order to see whether they might show recurrences. None was observed after a definite cure had been effected.

The observation of considerable enlargements of the spleens after repeated subcutaneous injections of concentrated spleen extract suggested the idea that these spleens might contain active substances and that it would be of interest to prepare an extract from them.

For brevity's sake this extract will be called "healed spleen extract" in this paper.

Healed spleen extract is prepared in the following manner: The mice were killed with ether. The spleens were immediately removed, weighed, and frozen on blocks of dry ice. The frozen spleens were ground in the mortar with sand. Distilled water and chloroform were added at the following ratio: 5 grams of spleen was mixed with 50 cubic centimeters of water and 5 cubic centimeters of chloroform. The mixture was shaken for about 8 hours in a shaking machine and kept in an ice-box for at least 48 hours. The proteins were precipitated with 10 per cent trichloracetic acid. The mixture was centrifuged for about 15 minutes at the rate of 3000 revolutions per minute. The supernatant light yellowish fluid was passed through a filter paper and con-

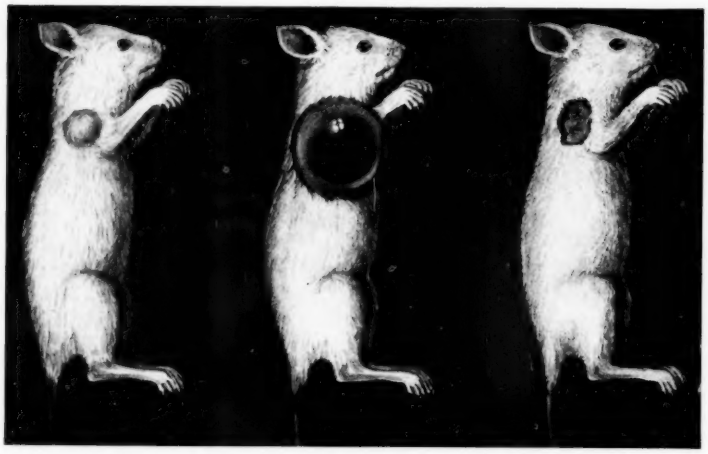

Fig. 3. Mouse 132. Treatment started March 26, 1939; healed July 10.

centrated at 60 degrees *in vacuo*, until 1 cubic centimeter of the extract represented 1 gram of fresh spleen. The acid extract was neutralized with sodium hydroxide with litmus paper as indicator and filtrated through a Seitz filter No. 3. Merthiolate (1:200000) was added and the extract was collected in sterile ampoules.

ACTION OF HEALED SPLEEN EXTRACT ON SARCOMA 180

The number of injections necessary to effect a cure varied according to the period which had elapsed between the cure of the mice whose spleens were used for the extract and the preparations of healed spleen extract. In the earlier experiments the spleen extract had been obtained from animals which had been cured for 4 or 5 months. When this extract

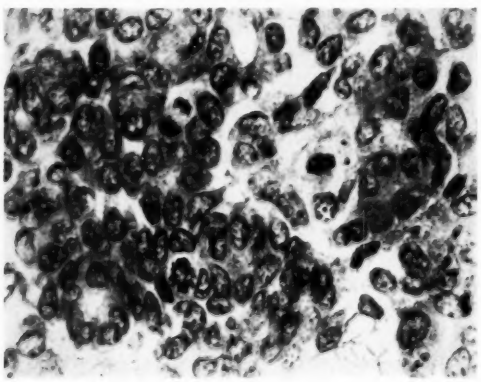

Fig. 4. Photomicrograph, mouse 132, showing adenocarcinoma, strain A. Several mitoses are evident.

TABLE I.—SARCOMA 180 TREATED WITH INTRAVENOUS INJECTIONS OF HEALED SPLEEN EXTRACT

No. of set	Number of mice	Healed	Died with tumors
222	9	8	1
230	9	7	2
231	9	5	4
426/427	14	7	7
500	9	5	4
532	7	7	0
	57	39 (68%)	18

TABLE II.—CONTROLS. SARCOMA 180

Set passage	No. of mice	Healed	Dead
226/29			
48 (oxserum)	29	2	27
233/34			
50	20	1	19
426/427			
76	10	2	8
500			
86	10	3	7
528/32			
88	18	0	18
	87	8	79

The regressions were 9 per cent.

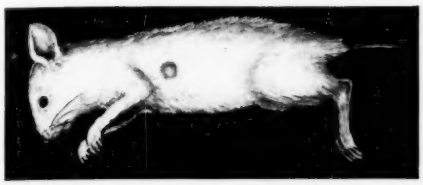

Fig. 5. Mouse 134. Treatment started April 6, 1939; healed April 21, 1939.

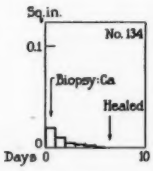

Fig. 6. Mouse 134. Five intravenous and 1 intraperitoneal injections of healed spleen extract were given.

was used, two or three intravenous injections of healed spleen extract were sufficient to effect a cure of sarcoma 180. In our more recent experiments we were forced to use the spleens a few weeks after the cure had been effected, in order to repeat the experiments on a larger scale. In this group of mice, 7 to 8 intravenous injections were needed instead of 2 or 3.

It is possible that the difference in the efficacy of the extract taken from animals cured recently or cured many months ago may be due to the gradual accumulation of "antibodies" in the spleen during a period of months following the disappearance of the tumors. It is well known that the production of antibodies may reach its highest peak after a few months.

Statistical data of the effect of healed spleen extract on sarcoma 180 are given in Tables I and II.

The effect of intravenous injections of healed spleen extract on sarcoma 180 differed very materially from the changes caused in these tumors following subcutaneous injections of concentrated beef spleen extract. The changes caused by the latter extract were described in detail in the previous paper (7). For this reason we shall refer to them here very briefly. In the main, four different stages were observed: (1) hemorrhage, (2) cavity, (3) scab, and (4) cure.

The manifestations of a specific action of the healed spleen extract when used intravenously were entirely different. The growth of the tumor was not inhibited after the first injection, in fact the size of the tumor increased for awhile. However, it stopped growing after a few injections.

The hemorrhage which has been described after beef spleen extract therapy was never

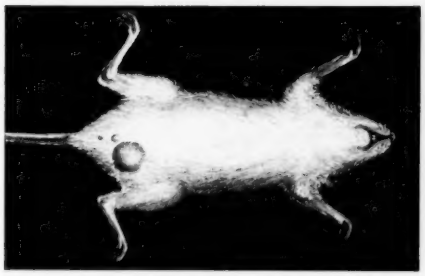

Fig. 7. Mouse 143. Treatment started April 13, 1939; healed April 29, 1939. Died June 5, 1939. Postmortem and microscopic examinations negative for carcinoma.

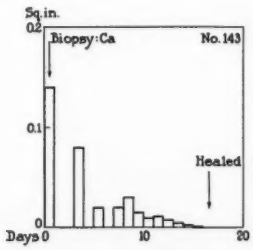

Fig. 8. Mouse 143. Twelve intravenous injections of healed spleen extract were given.

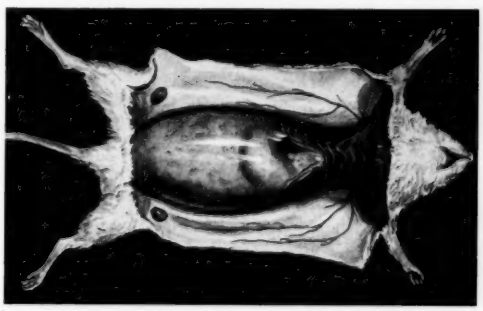

Fig. 9. Postmortem findings in mouse 143. Tumor has completely disappeared. Two inflammatory lymph nodes are present.

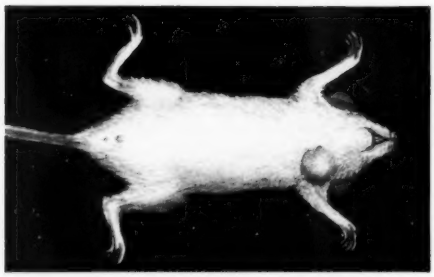

Fig. 10. Mouse 147. Treatment started April 26, 1939; healed May 19, 1939.

observed following the intravenous injection of healed spleen extract. As pointed out (7) this hemorrhage occurs in the majority of the cases a few hours after the first subcutaneous injection of concentrated beef spleen extract. On the other hand following the intravenous use of healed spleen extract the tumor (sarcoma 180) gradually dries up and shows central necrosis. This necrosis differs from the necrosis which is observed in untreated tumors. While the necrosis always stays centrally in the controls and the tumor continues to grow in the periphery, in animals treated with healed spleen extract the central necrosis breaks through the encircling tumor, resulting in mummification of the entire tumor. A sharp demarcation against the surrounding healthy tissue is noted. Gradually

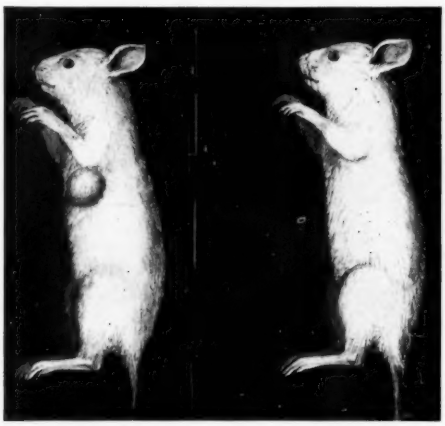

Fig. 12. Mouse 153. Treatment started April 27, 1939; healed May 19, 1939.

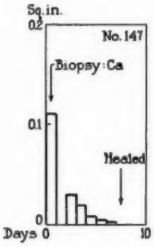

Fig. 11. Mouse 147. Six intravenous injections of healed spleen extract were given.

the mummified area is sequestrated. The raw aseptic area, which remains exposed after the exclusion of the mummified mass, epithelializes rapidly, and leaves no trace of tumor or inflammation.

During this process of restoration the general condition of the mice improves materially. They gain in weight and look like normal and healthy animals. After they are healed, the tumor does not recur. We have observed these animals for many months and have never discovered a recurrence.

We then compared the results obtained with healed spleen extract with spleen extracts prepared identically but obtained from untreated normal mice and from normal mice which had been treated previously with 14 subcutaneous injections of concentrated beef spleen extract. These extracts did not influence at all the growth of the tumors (Table III).

In the sarcoma 180 group treated with intravenous injections of healed spleen extract 39 were cured among 57 treated animals (Table I). While the number is limited the results were absolutely uniform and we there-

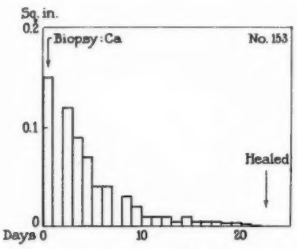

Fig. 13. Mouse 153. Thirteen intravenous and 1 intraperitoneal injections of healed spleen extract were given.

Fig. 14. Mouse 155. Treatment started April 28, 1939; healed May 18, 1939.

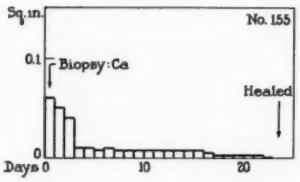

Fig. 15. Mouse 155. Eighteen intravenous injections of purified concentrated spleen extract were given.

fore were satisfied with the results on a relatively small number of experiments. This course was also dictated by the obvious necessity to save the valuable material for the more important experiments with spontaneous mouse tumors.

A comparison of the effect of healed spleen extract with that of concentrated spleen extract shows a higher incidence of cures as well as a distinct shortening of the period of treatment. The superior effect of healed spleen extract might be attributed to the intravenous route of injection which could not be used with the concentrated beef spleen extract because of its thrombosing action. However, this is certainly not the deciding factor because healed spleen extract is only 1/100 of the concentration of the beef spleen extract. It might be suggested that the greater efficacy of healed spleen extract is due to a biological concentration and that by combination of the two principles the activity of healed spleen extract may be improved.

Furthermore the difference in the response of the tumors to treatment with healed spleen extract on the one side and with concentrated beef spleen extract on the other side suggests differences in the active principles of both extracts. In the previous experiments with concentrated beef spleen extracts disappearance of the tumor was invariably initiated by hemorrhage into the tumor. In contrast healed spleen extract injections effected a disappearance of the tumor without hemorrhage. Hemorrhage is therefore not an essential prerequisite of tumor cure. This observation is paralleled by our experiments with histamine. Sometimes histamine causes hemorrhage into the tumor without a subsequent cure, whereas in other cases a cure is effected without preliminary hemorrhage. In fact, of the 25 mice healed by histamine, only 11 showed initial hemorrhage. While differences in the action and activity of concentrated beef spleen extract and healed spleen extract are obvious it is most probable that the active principle of both extracts is derived from the spleen as the main reticulo-endothelial organ of the body. This conclusion seems justified because

Fig. 16. Mouse 157. Treatment started April 28, 1939; healed July 14, 1939.

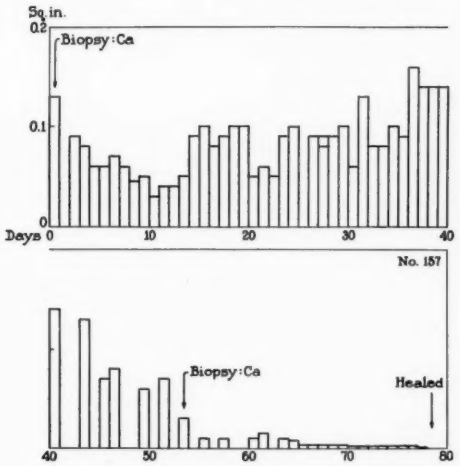

Fig. 17. Mouse 157. There were given 43 intravenous and 3 intraperitoneal injections of purified concentrated spleen extract.

Fig. 18. Control as of April 12, 1939, at left, and as of July 21, 2 views.

of the negative results with the injections of some other organ extracts and the not very impressive percentage of cures with the use of liver extracts from healed mice (Table IV).

The fundamental importance of the spleen as shown in these experiments suggests the attempt to influence spontaneous tumors by transplanting the spleen of healed mice intraperitoneally.

Finally attention must be called to the interesting observation that the only effective spleen extract was that prepared from mice which had been cured of the transplanted tumor by the injection of concentrated beef spleen extract. The spleen of mice with spontaneous regressions does not seem to have any effect on transplanted or spontaneous tumors. In order to obtain successful results from intravenous injections of healed spleen extract in a large percentage of transplanted mouse sarcoma 180 the following sequence is required: (1) growth of sarcoma 180 in mice to moderate size; (2) subcutaneous injections of 0.5 cubic centimeter of concentrated spleen extract until tumor disappears, an average of 14 injections being required; (3) preparation of extract of spleen according to directions given herein.

Further experiments were made in order to test the effect of histamine and of extracts of organs of healed mice other than spleen. Histamine[1] was injected in single doses of 0.5

[1]Furnished by Hoffmann La Roche Co.

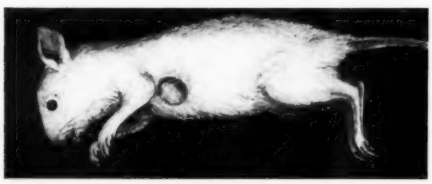

Fig. 19. Mouse 148. Treatment started April 20, 1939; died June 16, 1939.

Fig. 20. Postmortem findings mouse 148. The rest of the malignant tumor. About the same character as the original tumor.

TABLE III.—SARCOMA 180 TREATED INTRAVENOUSLY (A) WITH SPLEEN EXTRACT FROM NORMAL MICE, AND (B) WITH SPLEEN EXTRACT FROM MICE WHICH HAD RECEIVED 14 SUBCUTANEOUS INJECTIONS OF CONCENTRATED SPLEEN EXTRACT

Number of set	Number of mice	Healed	Died with tumors
(a) 238.....	10	0	10
(b) 239.....	10	0	10

cubic centimeter of 0.5—1.5 per cent solution. The organ extracts were obtained from the same animals as the spleen extract and prepared in an identical manner as the healed spleen extract. The single dose was also the same. Injections of histamine as well as organ extracts were continued until either the tumors had disappeared or the animals had died. The results of these experiments are summarized in Table IV.

TABLE IV.—RESULTS OF TREATMENT IN 398 SARCOMA 180 MICE WITH HISTAMINE AND DIFFERENT EXTRACTS FROM ORGANS OTHER THAN SPLEEN. "HEALED MICE" COMPRISE SARCOMA 180 MICE IN WHICH PREVIOUS SUBCUTANEOUS INJECTIONS OF CONCENTRATED SPLEEN EXTRACT HAD EFFECTED A CURE

Number of mice	Treated with	Route of injection	Healed	Died	Healed per cent
175	Histamine	Subcutaneous	25	150	15
74	Liver extract from "healed mice"	Intravenous	16	58	21
69	Liver extract from "healed mice"	Subcutaneous	12	57	17
40	Peritoneum extract from "healed mice"	Subcutaneous	4	36	10
10	Omentum extract from "healed mice"	Subcutaneous	0	10	0
10	Thymus extract from "healed mice"	Subcutaneous	0	10	0
10	Liver extract from spontaneous regressions	Subcutaneous	0	10	0
10	Peritoneum extracts from spontaneous regressions	Subcutaneous	0	10	0

ACTION OF DIFFERENT SPLEEN EXTRACTS ON SPONTANEOUS MALIGNANT TUMORS IN MICE (ADENOCARCINOMAS OF THE BREAST)

It seemed of considerable importance to test the action of chemically or biologically concentrated spleen extracts on spontaneous adenocarcinomas in mice. The animals used (strain A) were received from the Jackson Memorial Laboratory in Bar Harbor, Maine. We wish to express our appreciation to Dr. C. C. Little and his staff for their splendid cooperation.

When we began to study the effect of spleen extract on spontaneous tumors it soon became evident that the concentrated spleen extract which had given very good results when injected subcutaneously to sarcoma 180 mice, did not cure spontaneous carcinomas. One of the reasons was that, in order to effect a cure, we needed intravenous or intraperitoneal injections; subcutaneous applications of the extract did not act on these tumors. We soon found that we could not use this extract intravenously as it caused (after 2 or 3 injections) a venous thrombosis which made further treatment impossible.

PREPARATION OF SPLEEN EXTRACT

We approached this problem from a number of different angles. We used not only healed spleen extract, but we used a spleen extract from a young bull which had received many subcutaneous tumor transplantations extending over many months. We call this extract (again, for brevity's sake) immune spleen extract.[1] Another extract which was used successfully in the cure of spontaneous tumors was the original concentrated spleen extract which had been purified in this laboratory. This purified concentrated spleen extract does not cause thrombosis of the veins. With this purified extract we were able to give 43 intravenous injections (animal 157). Thirty-three intravenous injections of healed spleen extract were given to animal 123.

We are reporting results with the following spleen extracts: (1) Healed spleen extract (H.S.E.) (1 gm. in 1 c.cm.); (2) immune spleen extract (I.S.E.) (1 gm. in 1 c.cm.); (3) purified concentrated spleen extract (P.S.E.) (100 gm. in 1 c.cm.).

The preparation of healed spleen extract was described in the chapter on transplanted tumors.

[1]We have found recently that Lumsden injected serum of a sheep many times inoculated with sterile suspension of cancer fragments directly into spontaneous cancer of mice. He observed extensive necrosis, and a cure in 10 per cent of the animals. The indirect intravenous approach failed to give results.

TABLE V.—REVIEW OF MATERIAL—SPONTANEOUS ADENOCARCINOMA OF BREAST[1]

Number of animals	Treatment	Healed	Reduced in size	Not influenced
38	H S E	13	14	11
14	I S E	5		9
33	P S E	7	11	15
Total 85		25	25	35
Per cent		30	30	40

[1]Last reading September 12, 1939.
H. S. E., healed spleen extract.
I. S. E., immune spleen extract.
P. S. E., purified concentrated spleen extract.

Immune spleen extract was prepared in the following way: The animal was killed and the spleen was removed immediately. Preparation and preservation of the extract was practically identical with that for healed spleen extract which was given in detail in the first chapter.

Purified concentrated spleen extract was prepared by dialysing the concentrated spleen extract. These experiments are still in their initial stage.

We have recently modified the preparation of these extracts. With these modified extracts we can give larger doses without causing toxic symptoms. However, we feel that before we are ready to standardize the technique and report it in detail, we should experiment with these extracts on a larger scale.

In order to avoid any possible misinterpretation in the proper classification of these tumors we have performed a biopsy in every case herein reported. We have included in our series typical adenocarcinomas only. All sections were submitted for diagnosis to our pathologist, Dr. Klemperer. Whenever the slightest doubt existed as to the malignant character of the tumor, the case was excluded.

EXPERIMENTS

We are presenting records of 25 mice which were cured of their malignant tumors (spontaneous adenocarcinomas of the breast) by treatment with spleen extracts. They represent 30 per cent of the treated animals. In another 30 per cent the tumors either had been arrested or they showed a marked diminution in size at the time of death (see, for instance, animal 148). Thus we were able to influence and retard tumor growth in 60 per cent of the animals. The 40 per cent of the animals remaining were resistant to treatment. The tumors continued to grow, as in the controls.

The comparatively small number of cured animals (25) may be explained in the following manner:

1. The Jackson Memorial Laboratory was not able to send us more than a few animals each week.

2. With our limited laboratory facilities we could not take care of a very large number of spontaneous tumors, as the treatment is not only time-consuming, but the animals require most careful individual attention.

Of the 25 animals 13 were cured by healed spleen extract, 5 by immune spleen extract and 7 by purified concentrated spleen extract.

The number and method of the injections (intravenous, intraperitoneal, or subcutaneous) and the nature of the extract are described with the individual pictures or in the appended case histories. In addition the diagrams give a very clear day by day picture of the influence of the spleen extract on the tumor growth.

The action of the extracts on spontaneous tumors differs from the action of concentrated spleen extract on sarcoma 180, which was described in the previous paper. The initial hemorrhage did not appear and the typical cavities were not observed. After an initial state of edema the tumors in the majority of cases began to shrink gradually without presenting any necrosis.

There seems to be a more or less definite relation between the size of the original tumors and the number of injections necessary to effect a cure. Small tumors may disappear completely after a few injections, whereas larger tumors may require 30 or more injections to obtain this result. Furthermore the response to treatment seems to depend somewhat on the character of the tumor. Hemorrhagic tumors seem to be more resistant than the unchanged tumors.

Spontaneous regressions in proved carcinomas are unknown. Upon inquiry Dr. Little stated that in these sets of mice they had never seen a spontaneous regression of a malignant tumor when proved by biopsy.

Since we have performed biopsies, we have learned to appreciate the great importance of this step, as about 20 per cent of the biopsies proved to be adenomas or borderline cases. Naturally they were excluded from our series though Haaland and others have claimed that they should be included in the group of malignant tumors. Without biopsies these animals would have been included in our material of malignant tumors as palpatory findings were identical with true carcinomas.

The injections were given intravenously or intraperitoneally. The extracts were injected intravenously into the dorsal or ventral vein in the tail of the mouse. The fact that up to 43 intravenous injections were given to one animal is the best proof that we have succeeded in removing most of the irritating substances from the extract.

Whenever we found it impossible to continue intravenous injections, the intraperitoneal approach was employed.

Naturally the dose for intravenous therapy varied somewhat with the nature of the different extracts. Thus the average dose for healed spleen extract was 0.15 cubic centimeter, whereas purified concentrated spleen extract could be given in slightly larger doses.

In the earlier work the injections caused some shock. With the purification of the extracts the severity of the shock has been lessened considerably, so that in many instances it is hardly noticeable (except with modified spleen extract which will require further detoxication).

Though the actual size of these tumors may appear rather small, they would correspond to a human tumor weighing about 150 grams—a tumor of considerable size.

The subcutaneous dose of 0.5 cubic centimeter of spleen extract corresponds to a human dose of 100 cubic centimeters. The intravenous dose of 0.1 cubic centimeter would correspond to a dose of 20 cubic centimeters.

The life expectancy of a mouse is about 2 years as compared with the human life expectancy of about 60 years. The mice begin to show the tumors when they are about 1 year and 3 months old, which corresponds to 40 years in the human. The analogy in the cancer age of the mouse (1 year and 3 months) and in the human (40 years and more) is of considerable interest.

TABLE VI.—DAYS ELAPSED SINCE SPONTANEOUS MALIGNANT TUMORS WERE HEALED[1]

	Animal no.	Healed since	No. of days
1	123	May 29, 1939	106
5	147	May 19	116
6	153	May 19	116
7	155	May 18	117
8	156	May 31	104
9	157	July 14	50
10	183	July 10	64
11	187	July 4	70
12	192	July 8	66
13	194	July 20	54
14	196	July 26	48
15	198	July 20	54
16	210	July 27	47
17	218	July 27	47
18	199	Aug. 7	36
19	207	Aug. 7	36
20	231	Aug. 3	40
21	224	Aug. 7	36
22	228	Aug. 14	29
23	238	Aug. 25	18
24	232	Sep. 10	3
25	234	Sep. 10	3

[1]Last reading September 12, 1939.
2. Animal 132 died July 10. Postmortem and microscopic examination showed no carcinoma.
3. Animal 134 died May 10. Postmortem and microscopic examination showed no carcinoma.
4. Animal 143 died June 5. Postmortem and microscopic examination showed no carcinoma.

It is evident from the illustrations that we have succeeded in curing not only small tumors but carcinomas of fairly good size. However, it should be pointed out that at present very large tumors do not respond to the treatment. We hope to overcome this difficulty by further improvements of the extracts.

We have tried to transplant intraperitoneally "healed" spleens (spleens obtained from sarcoma 180 mice which had been cured by treatment with concentrated spleen extract) to mice, bearing spontaneous tumors. However, as other observers had noticed before us, the toxicity of these transplants is so great that the mortality is prohibitive. Though in the surviving animals we had some promising

results, the numbers were too small to draw any conclusions.

An interesting observation is presented by animal 148 A and B. The moderate size tumor had apparently disappeared after 8 intravenous and intraperitoneal injections of healed spleen extract. Further injections were stopped as palpatory findings failed to show evidence of tumor. When the animal died 6 weeks later, a small rest of tumor was observed (animal 148 B). It is very probable that if we had continued intravenous injections for some time after the disappearance of palpatory findings, the postmortem findings would have been completely negative, as in animals 132, 143 A and B, and 134.

BRIEF ABSTRACTS OF CASES OF CURED SPONTANEOUS BREAST ADENOCARCINOMAS IN MICE

CASE 1. Mouse No. 123. J. M. L.[1] Biopsy specimen showed carcinoma. Treatment was started February 18, 1939; healed May 29. Thirty-three intravenous and 19 intraperitoneal injections of healed spleen extract were given. Black tumor. Biopsy specimen was taken after 34 days. Our first biopsy. Animal was cured in 78 days. There were occasional increases in size (see diagram) probably partly due to slight errors in measuring. (Tumors in the distal half of the body are more difficult to influence than those in the proximal half.) (Figs. 1 and 2.)

CASE 2. Mouse No. 132. J. M. L. Biopsy specimen showed carcinoma. Treatment was started March 26, 1939 and animal died July 10. Nineteen intravenous, 16 intraperitoneal, and 8 subcutaneous injections of healed spleen extract were given. Postmortem and microscopic examinations were negative for carcinoma. (Figs. 3 and 4.)

CASE 3. Mouse No. 134. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 6, 1939; healed April 21. Five intravenous and 1 intraperitoneal injection of healed spleen extract were given. Animal died May 10. Postmortem and microscopic examinations were negative for carcinoma. (Figs. 5 and 6.)

CASE 4. Mouse No. 143 A and B. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 13, 1939; healed April 29. Twelve intravenous injections of healed spleen extract were given (Figs. 7 and 8). Animal died June 5. Microscopic and postmortem examinations were negative for carcinoma but two small inflammatory glands were noted (Fig. 9).

CASE 5. Mouse No. 147. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 26, 1939; healed May 19. Six intravenous injections of healed spleen extract were given (Figs. 10 and 11).

CASE 6. Mouse No. 153. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 27, 1939; healed May 19. Thirteen intravenous and 1 intraperitoneal injections of healed spleen extract were given (Figs. 12 and 13).

CASE 7. Mouse No. 155. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 28, 1939; healed May 18. Eighteen intravenous injections of purified concentrated spleen extract were given (Figs. 14 and 15).

CASE 8. Mouse No. 156. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 28, 1939; healed May 31. Twenty-five intravenous injections of purified concentrated spleen extract were given. A double tumor was present, one of which was a cystic mass. Biopsy specimen was taken from the solid tumor only.

CASE 9. Mouse No. 157. J. M. L. Biopsy specimen showed carcinoma. Treatment was started April 28, 1939; healed July 14. Forty-three intravenous and 3 intraperitoneal injections of purified concentrated spleen extract were given (Figs. 16 and 17).

CASE 10. Mouse No. 183. J. M. L. Biopsy specimen showed carcinoma. Treatment was started June 15, 1939; healed July 10. Two intravenous, 11 intraperitoneal, and 3 subcutaneous injections of immune spleen extract were given. Dorsal tumor.

CASE 11. Mouse No. 187. J. M. L. Biopsy specimen showed carcinoma. Treatment was started June 22, 1939; healed July 4. Ten intravenous injections of modified healed spleen extract were given.

CASE 12. Mouse No. 192. J. M. L. Biopsy specimen showed carcinoma. Treatment started June 29, 1939; healed July 8. Four intravenous and 2 intraperitoneal injections of immune spleen extract were given.

CASE 13. Mouse No. 194. J. M. L. Biopsy specimen showed carcinoma. Treatment was started June 29, 1939; healed July 20. Sixteen intravenous injections of modified immune spleen extract were given since July 20, at which time a biopsy was taken. This second biopsy, taken to ascertain whether a small palpatory mass was malignant, proved negative. Microscopic examination of this biopsy was negative for carcinoma.

CASE 14. Mouse No. 196. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 8, 1939; healed July 26. Small tumor. Sixteen intravenous injections of modified healed spleen extract were given.

CASE 15. Mouse No. 198. J. M. L. Biopsy specimen showed carcinoma. Large black posterior tumor. Treatment started July 8, 1939; healed July 20. Seven intravenous and 1 intraperitoneal injections of healed spleen extract were given.

CASE 16. Mouse No. 210. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 13, 1939; healed July 27. Twelve intravenous injections of modified healed spleen extract were given.

[1] J.M.L., Jackson Memorial Laboratory.

CASE 17. Mouse No. 218. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 20, 1939; healed July 27. Five intravenous injections of immune spleen extract were given.

CASE 18. Mouse No. 199. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 8, 1939; healed August 7. Twenty-three intravenous injections of modified healed spleen extract were given. A second tumor appeared on July 11, the third day after injection, and disappeared on July 31, the twentieth day after injection. Twenty-three intravenous injections of healed spleen extract were given.

CASE 19. Mouse No. 207. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 12, 1939; healed August 7. Ten intravenous injections of healed spleen extract were given. On September 1 a small recurrence was noted. Biopsy examination revealed an adenocarcinoma. This responded to treatment which consisted of 10 intravenous injections. On September 10 a complete disappearance of the recurrent tumor had taken place.

CASE 20. Mouse No. 231. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 27, 1939; healed August 3. Six intravenous injections of modified spleen extract were given.

CASE 21. Mouse No. 224. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 27, 1939; healed August 7. Seven intravenous injections of modified immune spleen extract were given.

CASE 22. Mouse No. 228. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 27, 1939; healed August 15. Twelve intravenous injections of modified healed spleen extract were given.

CASE 23. Mouse No. 238. J. M. L. Biopsy specimen showed carcinoma. Treatment was started August 2, 1939; healed August 25. Fourteen intravenous injections of modified spleen extract were given.

CASE 24. Mouse No. 232. J. M. L. Biopsy specimen showed carcinoma. Treatment was started July 27, 1939; healed September 10. Thirty-five intravenous injections of purified concentrated spleen extract were given.

CASE 25.[1] Mouse No. 234. J. M. L. Biopsy specimen showed carcinoma. Treatment was started August 3, 1939; healed September 10. Twenty-four intravenous injections of purified concentrated spleen extract were given.

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[1] Correction (January 1, 1940): Since the presentation of the paper we have noticed recurrences in 8 mice (Nos. 156, 183, 187, 192, 196, 198, 224, 231). We feel that these recurrences may be explained by the fact that treatment was discontinued immediately after the tumor had disappeared. In view of the scarcity of the supply (especially healed spleen extract) we tried to give as few injections as possible. Since we observed these recurrences, we have continued to inject the healed animals once or twice a week. No further recurrences have occurred since we followed this procedure. Animals 123, 194, 199, 207, 210, 218, 228, 238, 232 and 234 are alive and well today (3½ to 7 months). One healed mouse (No. 153) was missed on October 3. In 6 mice (Nos. 132, 134, 143, 147, 155, 157) which have died since they were healed, the postmortem and microscopic examination failed to show any sign of carcinoma. We have 23 additional healed mice not reported in this paper. Thus the total of healed mice amounts to 39 animals.

PELVIC PERITONITIS IN FEMALE INFANTS AND CHILDREN

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IN a specialized study of genital pathology in 371 female infants and children, considerable opportunity has been offered to observe instances of infection of the intrapelvic genitalia. Out of the entire group, 259 patients have presented frank vaginal infections of which 53 per cent were proved positive for gonorrhea, and an additional 33 per cent were considered highly suggestive of a gonorrheal origin. With the potentiality of ascending infection constantly in mind, we have been able to make observations which should be of value in this altogether too obscure field. Nine instances of proved or highly probable pelvic infection have been studied. We will discuss in order: (1) Important points of difference in methods of examination in the immature as opposed to the mature female; (2) sources and pathways of infection; (3) pathology; (4) symptoms, signs and diagnosis; (5) pertinent data in 9 personal cases.

IMPORTANT POINTS OF DIFFERENCE IN METHODS OF EXAMINATION OF THE IMMATURE AS OPPOSED TO THE MATURE FEMALE

The importance of precision and the knowledge gained by experience cannot be overestimated. It is essential that the specialist in this field be intimately acquainted with the anatomical and physiological differentials between immature and mature individuals. Failure to appreciate such factors will result in unnecessary pain and fright, and may serve as the nucleus for a later sex fear complex of alarming proportions. Whereas one may easily overemphasize the potentiality of such manipulations in female infants and small girls, the specialist may not ride rough shod into the management of these conditions without the possibility of serious repercussions.

Precision technique relative to the lower genital tract has been described and illustrated in another communication.[1] In connection with disease of the intrapelvic genitalia, certain points in relation to vaginal, rectal, and abdominal examination should be reemphasized here.

Digital, vaginal examination may be accomplished without serious difficulty in a considerable number of girls between the ages of 10 and 14 years. In the interest of thoroughness a careful attempt to conduct such an examination should be made in each instance. If the child becomes alarmed, or the distress is too great, immediate recourse should be had to rectal examination. If rectal examination does not yield sufficiently helpful findings, vaginal examination should be done under anesthesia. Satisfactory anesthesia for such a purpose presupposes complete relaxation. Ethylene has been found in general the most satisfactory anesthetic for this purpose. The operator must at no time forget that anesthesia does not protect the child from potential harm contingent upon rough manipulation. Particularly in the case of suspected pelvic infection with possible abscess formation should extreme care be used not to traumatize the delicate and more accessible structures of the immature pelvis.

The accompanying schema (Fig. 1) illustrates the different spacial relationships, positions, and relative sizes of organs in the immature and the mature individual. Whereas the structures may not be more easily or perhaps even *as* easily identified due to the thickness and lack of puerperal differentiation of the pelvic floor in these young patients, it must be borne in mind that the examining finger or instrument is relatively considerably longer and larger; pelvic structures are very much more accessible to pressure and digital trauma; and a large examining finger at full insertion in an immature vagina may offer a serious threat even to normal pelvic genitalia. This

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Read before the meeting of the North Pacific Surgical Association, Victoria, B. C., November 17 and 18, 1939.

[1] Schauffler, Goodrich C. Special methods of examination and technic of treatment in female infants and children. Obstetrics, Gynecology, Urology, and Proctology. (In press.)

warning applies to rectal as well as to vaginal manipulation and should carry with it the concept that anesthesia may create a false sense of security on the part of the examiner.

Points which may be elucidated by vaginal or rectal examination or both are roughly as follows: (1) a normal or abnormal general habitus; (2) pelvic tenderness and its localization—unanesthetized patient; (3) pelvic masses and their location; (4) cul-de-sac or rectal wall bulging with or without fluctuation (Fig. 2). Naturally the contribution of the abdominal hand to this examination is important.

Reasons why the examining finger in the child or infant may determine less than in the adult are: The pelvic floor in the child is thick, muscular and undifferentiated by the physiological factors or the mechanical effects of childbearing. Structures clearly defined in the adult, such as the anterior or posterior cul-de-sac exist only as small somewhat indefinite indentations at the bottom of the pelvis. Structures to be examined are very much smaller and more closely packed. Thus, while these same structures are more accessible in so far as actual distance is concerned, tissue thickness, muscle, and fascial investments and the diminutive size of these structures create a definite disadvantage in examination of the immature individual.

Rectal examination we estimate as perhaps 30 per cent less efficient than vaginal examination in the adult. Whereas rectal examination may be easily and relatively painlessly performed, even in very small infants, there is little to alter this differential in relation to its diagnostic value. Rectal examination, however, will of necessity be more frequently used in children and infants, and the stereognostic education of the index finger is quite as important in this special department as it is elsewhere. The examiner specifically experienced in rectal examinations in female children should be, and is, able to determine more than his untrained contemporary. This factor should be borne clearly in mind in relation to consultation; the pediatrician may be more helpful in such a situation than the general surgeon or even the gynecologist.

Abdominal examination should be conducted with more than usual attention to the patient's reaction. Careless prodding will evoke voluntary spasm as a response either to a tickling reflex or to concern in regard to rough handling. In this connection the old expedient of immersing the child in a hot bath is extremely helpful. The relaxing effect of this procedure is surprising and highly gratifying. It should be used more often than it is.

SOURCES AND PATHWAYS OF PELVIC INFECTION

That the source of pelvic infection in immature females is nearly always *via* the genital tract from below is scarcely disputed. Very little reliable case material is available and conclusions must be to a considerable degree speculative. Exceptions, of course, may occur in cases of general sepsis or pyemia. Causative organisms are, in order of frequency, the gonococcus, pneumococcus, colon bacillus, streptococcus, and staphylococcus. Naturally, the genesis of gonorrheal pelvic disease can be consistently ascribed to the ascending route, *via* the mucous membranes. Interestingly enough a fairly high percentage of pelvic infection in these immature individuals is ascribed to the pneumococcus, and even more interesting is the fact that such infection is seldom via the blood stream, but nearly always by ascent through the lower genitalia. These deductions, by the way, are not speculative. Careful bacteriological study in several series of cases has established the presence of identical type pneumococci in the vagina and in the infected adnexa. Of course, pneumonia is generally an antecedent condition, yet pneumococcus pelvic peritonitis in female children is not infrequently recorded in the absence of discernible pneumococcus infection elsewhere. It is of particular interest that even when peritonitis from this organism occurs in the presence of pulmonary involvement, the adnexal infection is set up, in most instances, by migration from the lower genitalia. The condition is seldom noted in males.

Streptococcus pelvic peritonitis is an extreme rarity in the immature female—a fact which attests its almost complete dependency upon injury to the continuity of mucous membranes contingent upon pregnancy or abortion. Infection by the colon bacillus is uniformly the result of rupture of an infected appendix

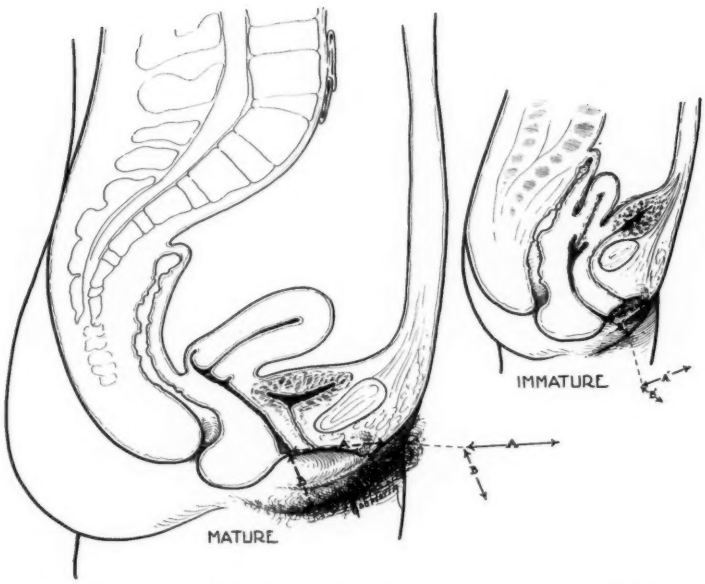

Fig. 1. Schema of sagittal sections of pelvis of, left, nulliparous adult and, right, 1 year old infant, to differentiate special relationships, size and relations of organs, thickness of septa (see also Fig. 2). Subtended lines *A* and *B* are for comparison with subtended lines *A'* and *B'* as denoting relative prominence of external genitalia, axis of vulvar orifice, protective distance to vaginal orifice, protective pubic hair. These factors indicate greater accessibility of immature genitalia to casual infection.

or some other primary pathological condition of the bowel (intussusception, etc.) and is secondary. Incidentally the offending appendix need not be pelvic in station to create the clinical picture of a primary pelvic peritonitis. Streptococcus and the even more rare staphylococcus infections have been almost without exception the accompaniments of general sepsis or pyemia. These infections, though rare, are extremely severe, and have furnished us with most of our postmortem pathological material.

The agency of a primary vaginitis as a focus for dissemination to pelvic structures is of especial interest. In several other communications relating specifically to primary infections of the immature vagina, we have insisted upon the importance of the mechanical conformation of this small organ, with its deep rugæ and crypts, and have repeatedly pointed to a reversal of rôles between the vagina and the endocervix, which in its turn is not the complicated, racemose, glandriddled structure noted in the adult. The immature cervix, in this instance, constitutes a relatively poor harbor of infection due to the late differentiation of its glandular elements (Fig. 3). These basic factors have been repeatedly illustrated and are borne out by the relative frequency of a primary vaginitis in the immature, and the relative infrequency of such an entity in the adult. Cervicitis on the other hand is a common entity in the adult and infrequent or virtually negligible in the young. Furthermore endocervicitis, when it does occur, we have noted only in older children (Fig. 4).

These observations offer a sound explanation for the extremely rare occurrence of upward extension of vaginal infection. The vagina, to be sure, harbors the infection, but the endocervix and endometrium are not fertile soil for mucous membrane retention or transmission of organisms.

Of our 266 patients with frank vaginal infections only 9 presented symptoms indica-

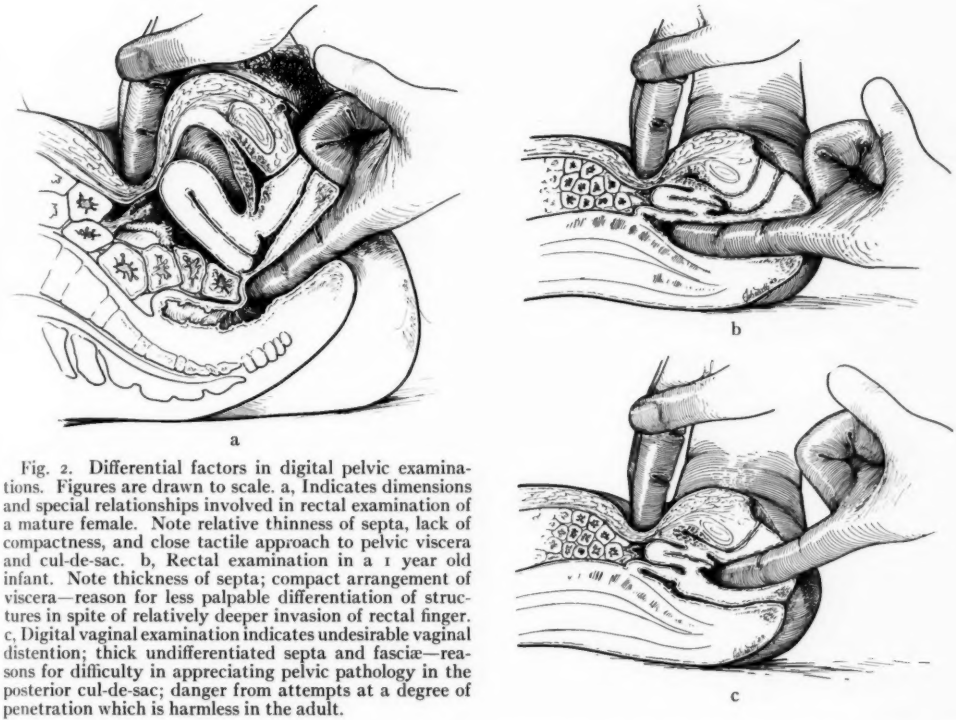

Fig. 2. Differential factors in digital pelvic examinations. Figures are drawn to scale. a, Indicates dimensions and special relationships involved in rectal examination of a mature female. Note relative thinness of septa, lack of compactness, and close tactile approach to pelvic viscera and cul-de-sac. b, Rectal examination in a 1 year old infant. Note thickness of septa; compact arrangement of viscera—reason for less palpable differentiation of structures in spite of relatively deeper invasion of rectal finger. c, Digital vaginal examination indicates undesirable vaginal distention; thick undifferentiated septa and fasciæ—reasons for difficulty in appreciating pelvic pathology in the posterior cul-de-sac; danger from attempts at a degree of penetration which is harmless in the adult.

tive of pelvic infection (0.034 per cent). In 3 of these the diagnosis was not definitely established. From this and other series, it is clear that the incidence of pelvic infection in immature females with lower genital gonorrhea is only a very small fraction of that noted in adults. Actual figures are available, but not entirely reliable. I regard the likelihood of ascending infection in these patients so lightly that I do not hesitate to tell worried parents that they need have little concern in this connection. Such a statement is eminently justified on the basis of our series, only 3 of our patients being sufficiently ill to warrant serious concern, and all at present are well and apparently functionally competent.

PATHOLOGY

Failing a clear concept of the status of immature pelvic viscera such as may be gained only at portmortem or at operation, one will scarcely appreciate what may be expected in infection of these organs. Outstanding differential factors from the adult are the very small size and lack of intricate differentiation of pelvic organs—uterus, tubes and ovaries, broad ligaments. It is not generally appreciated that the chief growth and adult differentiation of the uterus and endometrium occur very rapidly during a relatively short phase prepuberty and postpuberty. The uterus (Fig. 5) remains very small indeed, almost infantile in type, up to a matter of months prior to puberty. The endometrial glands are mere blunt truncated pits (Fig. 3). As for the tubes, they are very tiny structures indeed, their mucosa characterized by a much less complicated structure than that noted in the adult (Fig. 5). The ovaries also remain small and constitute very poor soil for retention of infection, there being no corpora hemorrhagica or cysts, to serve as harbors. We have, then, as the chief soil for intrapelvic infection, a young and resistant pelvic

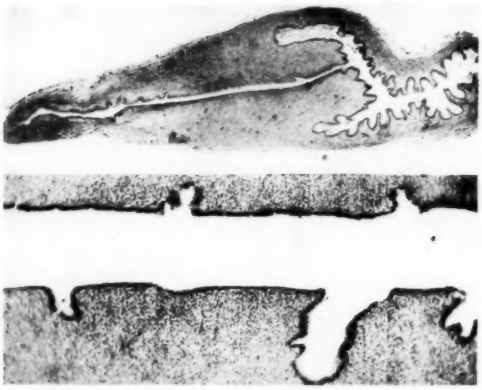

Fig. 3. Above, Block sagittal section of uterus and upper vagina at term. Note complicated plicæ of vaginal wall and, below, simple rudimentary type of cervical glandular structure (see also Fig. 4).

peritoneum. Naturally, therefore, pathology in this area does not follow adult patterns. Salpingitis *per se*, and particularly pyosalpinx and hydrosalpinx, as entities, are noted very seldom indeed, and then only in older children. The characteristic pathological development, and one which it is well to bear in mind in relation to differential diagnosis, follows the pattern of primary peritonitis elsewhere, with relatively frequent abscess formation—pointing above being rather more common than in the adult. Characteristic cul-de-sac fluctuation is seldom noted, due to the thickness and rigidity of the pelvic floor contingent upon lack of sexual and puerperal differentiation. In general, this pathology assumes, as we have said, the characteristics of peritonitis elsewhere in the abdomen—with fewer of the differential characteristics of progressive inflammatory disease of the adult adnexa.

SYMPTOMS, SIGNS, DIAGNOSIS

In this setting, simply to reiterate that the pathological condition in these cases follows the picture of a regional peritonitis should be sufficient. Unfortunately there are no striking differential points. The history, of course, is of the utmost importance. Vaginal discharge, past or present, particularly if of established gonorrheal origin, is of the greatest diagnostic assistance. I shall, however, cite an instance in which such a history seriously confused the true issue and was almost responsible for loss of life. Pneumonia, past or present, accompanied by lower abdominal symptoms, should be regarded with suspicion. A history suggestive of past or present appendix infection must be regarded seriously. The agency of severe injury from below—simple trauma or rape—should be obvious in such a setting. Severe pyelitis, especially with cystitis, may give a very confusing picture, since it frequently follows vaginal infection in a very deceptive sequence. Its rather definite characteristics, however, together with the urinary findings, should serve to differentiate it, ordinarily.

Temperature and white blood counts are, as would be expected, elevated beyond the usual adult recordings. We have observed the sedimentation rate in several of our cases, and note, in keeping with the general picture, less distinction between upper and lower abdominal conditions than in the adult female. This is perhaps due to the simpler and less vascularized condition of the immature pelvic genitalia. Sedimentation rate then is less helpful than in the adult in differentiating pelvic infections from other peritoneal involvements.

Because pelvic infection in the immature female is for the most part simple peritonitis, and because it does not cause the specific symptoms and signs attendant upon adult differentiation of pelvic structures, its differential diagnosis is much more difficult. On the other hand, such a differential virtually excludes certain confusing conditions which appear only in the adult, such as ectopic pregnancy, twisted pedicled or ruptured ovarian cyst, strangulated fibroid, and the like. Correct diagnosis as a rule at once establishes the presence of pelvic peritonitis. The question then arises as to its origin and its prognosis. If it can be shown by proof or sound inference to be of gonorrheal or pneumococcic origin, its management is conservative. If, on the other hand, it evolves from an infected appendix, it should in most instances be operable. The truly critical differential in this field is between primary pelvic peritonitis, demanding conservatism, and pelvic inflammation secondary

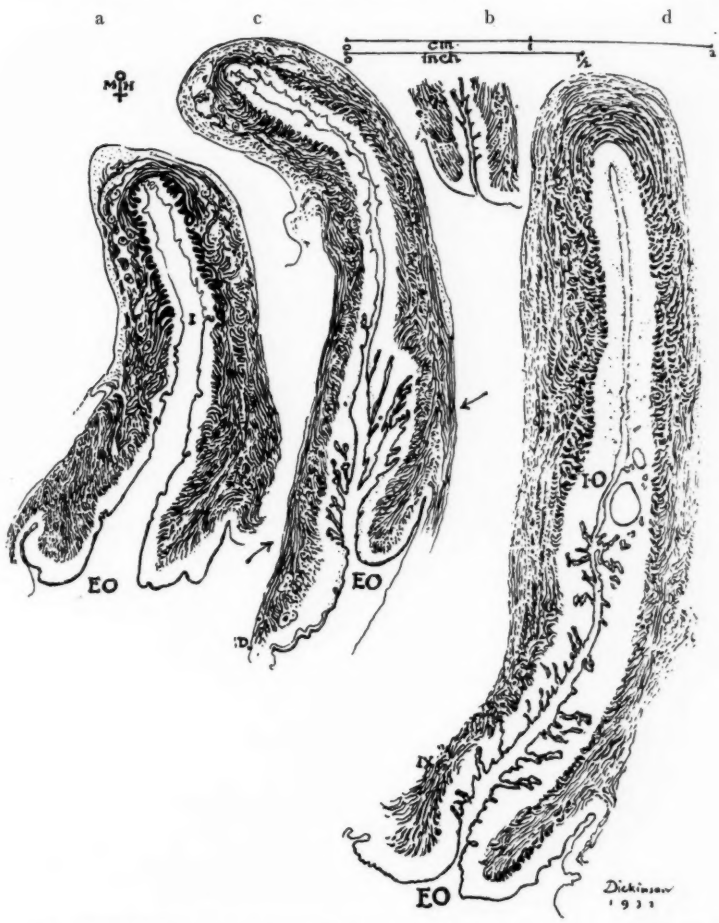

Fig. 4. Sagittal sections of immature uteri indicating relative simplicity of cervical glandular structure in children, complexity increasing with age. a, Two year old child—cervix runs from *I* internal os to *EO* external os; b, fourth year; c, ninth year, glands growing deeper, "uterus all cervix"; d, fifteenth year, not yet menstruating, thick lining of canal in c and d. (From Dickenson's *Human Sex Anatomy*, courtesy Williams and Wilkins, Baltimore.)

to an infected appendix, demanding early operation. *The very much higher incidence of appendicitis over pelvic infection in female children should be an important immediate consideration.*

Granted that a decision is difficult or impossible, the question between operative or conservative management should depend entirely upon the child's condition. Laparotomy for a suspected appendix such as was done upon one of our patients, even if in error, may be less harmful to the patient than failure to operate in the presence of a progressing appendix infection. On the other hand radical surgical interference in a belly which is at grips with a long ruptured appendix in a patient already seriously jeopardized, may be a grave error.

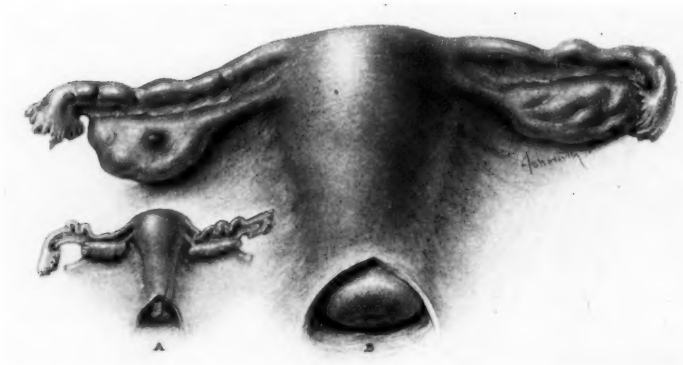

Fig. 5. Comparative sizes of uterus and adnexa in, a, stillborn infant at term (cervix hooked dorsad); b, mature adult. The importance of actual sizes of organs in relation both to diagnosis and pathology is indicated.

Drainage will occasionally be called for, and as I have pointed out, will more often be done abdominally than vaginally, since, for reasons indicated, pelvic abscess in the child less often points vaginally. In one of our patients abdominal drainage in the right lower quadrant constituted a life-saving measure. No hesitation should be felt in draining an available abscess through the rectum. This is frequently a very much more desirable procedure than clumsy attempts to invade a tiny cul-de-sac. Finally the resilience and recuperative powers of youth plus intelligent conservatism and supportive measures, may be heavily counted upon. Sulfanilamide has been employed in 2 of our cases, both gonorrheal; but we do not feel qualified to evaluate the results from this meager experience. The ability of children to withstand heavy concentrations of this drug should point to an excellent future for it in this condition.

Incident to operation upon infants and small children it is hardly necessary to call attention to certain factors of the utmost importance. It is difficult for the general surgeon who seldom operates upon these tiny patients to appreciate the differential involved, for example, in blood loss. Brunkow estimates that the loss of an ounce of blood in an 8 pound infant is equivalent to the loss of 18 ounces of blood in the robust adult. The strictest attention to hemostasis is, therefore, demanded. Heat and fluid loss from body surface appears to be very much more important in these little patients, the relative amount of body surface being very much greater. These and other factors assume critical importance due simply to geometric proportions. Here again specialized experience and meticulous technique may offer tremendous added safeguards. It is not to be denied that the surgeon's experience and skill in pediatric surgery may often constitute the differential factor between success and failure.

AUTHOR'S MATERIAL

For our own case material there is little point to a detailed presentation. We have seen 9 children in whom the diagnosis of primary pelvic peritonitis was made. The youngest was 3, the oldest, 14 years of age. Three of the diagnoses were not clearly established, 4 were definite, 2 were highly suggestive. Six were frankly gonorrheal. Our most serious case, in 1 of the patients who came to operation, was proved non-gonorrheal. One child developed a severe pelvic inflammatory reaction while under treatment by hypodermic theelin. Another had a quite typical pelvic flare-up in the presence of a gonorrheal vaginitis following a rectal injection of an antiseptic for associated gonorrheal proctitis. Three of these patients could at no time be demonstrated to be infected with gonorrhea. Two patients were suspected of having mild pneumococcic peritonitis, but

were not bacteriologically studied. Two were operated upon.

Two cases should be briefly described as pointing a moral.

CASE REPORTS

CASE 1. A small girl, aged 7 years, was operated upon for suspected appendicitis (by the surgical service). Only peri-appendicitis was present. Pelvic structures were visibly congested. There was considerable free fluid in the cul-de-sac and the picture was much more suggestive of a mild pelvic inflammatory reaction. A frank vaginitis with positive smears for gonorrhea was demonstrated *after* operation. Recovery was uneventful.

CASE 2. A child, aged 12 years, who had been treated in my special clinic for gonorrheal vaginitis several years before, entered the surgical service with a textbook history of acute appendicitis. There were no symptoms of vaginitis and smears were negative on admission. However, the surgical aspect of the case was neglected and the child was treated conservatively for a suspected pelvic inflammatory condition. An abscess developed in the right lower abdomen and pelvis which was later drained of large amounts of colon bacillus pus through a small right rectus incision. This child very nearly died. The surgical service at the present time is inclined to agree with us that infection with rupture of the appendix was overlooked because of a past history of gonorrheal vaginitis.

These 2 cases appear to justify the conclusions which we have already formulated, namely: With the patient in good condition, less harm may be done by a mistaken operation for suspected appendicitis than by deferring operation under a mistaken diagnosis of primary gonorrheal pelvic inflammation. It should be borne in mind that appendicitis in children is common, primary pelvic infection very uncommon indeed.

SUMMARY

1. Data from the records of 371 instances of female genital pathology in children and infants have been sifted for material in relation to primary pelvic infection. In 259 cases the original condition centered about a frank vaginal infection, the majority gonorrheal.

2. Important points of difference in methods of examination of the immature as opposed to the mature female are considered. Vaginal, rectal, and abdominal examinations are discussed in detail.

3. Sources of pelvic infection are considered. By far the most frequent route is thought to be by ascent from an infected vaginal tract. The gonococcus and the pneumococcus, the organisms most frequently involved, invade almost always in this manner. Secondary infection from the appendix is not infrequent, and is always serious.

4. Lack of adult and puerperal differentiation accounts for a tendency toward simple peritonitis, the ordinary sequence of involvements in the adult being rarely noted. The pathology is essentially that of pelvic peritonitis.

5. Differential diagnosis is briefly considered. Appendix infections and pyelocystitis are the most frequent confusing factors.

6. The very much higher incidence of appendicitis over primary pelvic infections in female children is a most important immediate consideration. The decision relative to operation is frequently crucial.

7. Pertinent data from 9 personal observations of actual or highly suggestive primary pelvic infection are reviewed.

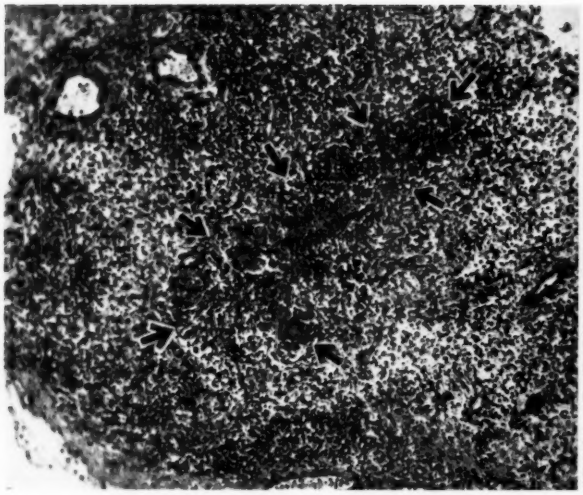

Fig. 1. Granulation tissue from a tuberculous anal fistula. In the background of young fibroblasts and capillaries a long ovoid tubercle (indicated by arrows) is seen with a Langhans' giant cell on its border. ×85.

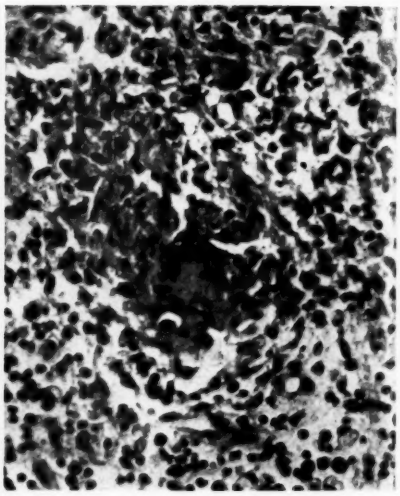

Fig. 2. Same tubercle as in Figure 1. Giant cell shows peripheral arrangement of nuclei in horseshoe formation. Epithelioid cells, many lymphocytes, and occasional plasma cells and polymorphonuclear leucocytes ×300.

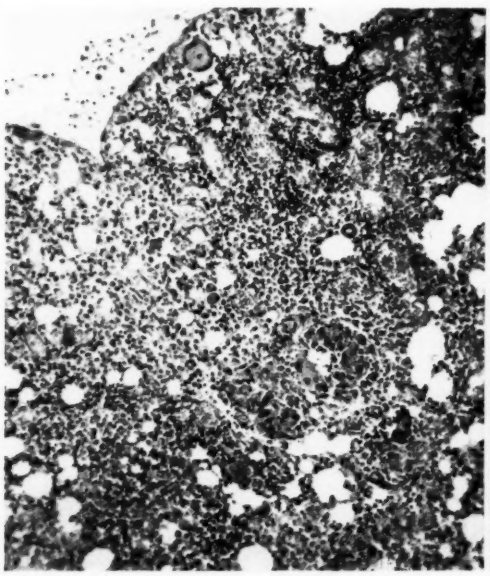

Fig. 3. Granulation tissue from another tuberculous anal fistula. A small tubercle consisting largely of "epithelioid" and other monocytic cells and an atypical giant cell at its center. Many empty capillaries are seen. ×87.5.

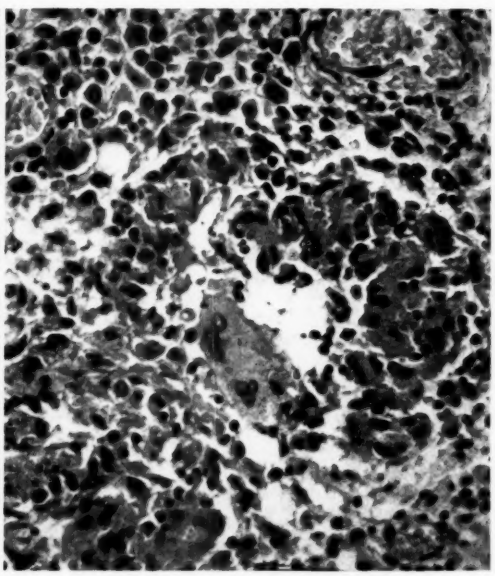

Fig. 4. Same tubercle as in Figure 3. Epithelioid cells large and very distinct. A discrete dense collection of such cells in round or oval arrangement is strongly presumptuous of tubercle formation. The atypical giant cell is almost pathognomonic. In such a specimen a search will almost invariably reveal other more typical cells. ×295.

Tuberculous Anal Abscess and Fistula Criteria for Diagnosis.—Clement L. Martin and Henry C. Sweany.

TUBERCULOUS ANAL ABSCESS AND FISTULA CRITERIA FOR DIAGNOSIS

CLEMENT L. MARTIN, M.D., F.A.C.S., and HENRY C. SWEANY, M.D., F.A.C.P., Chicago, Illinois

A MORE general agreement on the criteria for diagnosis of tuberculous anal fistula is necessary. What is a tuberculous anal fistula, or when is an anal fistula tuberculous? This question merits an exact answer, not only for the patient's sake but also if reported statistics are to be of value, informed opinion as to the criteria should be in agreement.

It is a well known and, we believe, a generally accepted fact that a clinical diagnosis founded only on the gross pathology is absolutely undependable even though the opinion is that of one thoroughly conversant with the lesion. Not so well known and accepted is the real value of histopathological examination of suspected tissue and the limited reliability of guinea-pig inoculation with preparations of tissue excised from the fistulous tract or abscess cavity wall.

The proponents of guinea-pig inoculation as the most valuable means of making a diagnosis of tuberculosis in peri-anal inflammatory and suppurative disease, particularly abscess and fistula, in most instances accept their findings at face value. The fact is there is a large element of error here which is either unknown or overlooked.

In a study of 200 patients with pulmonary tuberculosis which we recently reported[1] it was determined that in 30 per cent there are not only viable but virulent tubercle bacilli present in the lower sigmoid and rectum. In patients having anorectal abscess or fistula 34 per cent had such tubercle bacilli present in the last foot of the bowel within an hour after an enema.

Thus in a third of the cases tubercle bacilli could enter the rectal or anal orifice (the internal opening) of the fistulous tract. So whether the fistula was a tuberculous process or not, tubercle bacilli might be present in it in a third of the cases. This large margin of possible error is not considered in any article in the current literature on the subject. One reason for this is that so little work had previously been done to determine the incidence of viable tubercle bacilli in the terminal bowel.

It is absolutely impossible to wash or treat tissue excised or curetted from these fistulous tracts in any way known to bacteriologists so that a contamination from viable tubercle bacilli can be positively excluded. Excluding contamination of tissue by the tubercle bacilli in these cases is just as difficult as really sterilizing the skin of the hands. It follows that the use of cultural or inoculation methods to determine the character of a fistula in patients with an active pulmonary tuberculosis is rather fruitless as the findings must be qualified by a 30 per cent possibility of error. After positive culture or guinea pig inoculation of tissue from the fistula the pathologist can accurately only state: "The culture or guinea pig test is positive but it may be wrong as far as determining whether this is a tuberculous fistula or not in at least a third of the cases."

The chief difficulty arises in knowing what to call tuberculous granulation tissue. There is little question about the tissue with capillaries, fibroblasts, monocytes and typical horseshoe-shaped Langhans' giant cells. When the tissue is atypical there arises the most difficulty. Upon analysis, however, it is not the problem it may seem. In the first place, most non-specific abscesses are composed of acute inflammatory tissue which may be eliminated promptly. The tissue of a chronic or subacute nature may or may not have tuberculous granulation tissue. In such borderline cases the clinical appearance of the lesions, the clinical history, and the bacteriological findings will help to supplement any questionable lesions.

In Figure 1 is shown chronic granulation tissue with a predominance of monocytic cells

From the City of Chicago Municipal Tuberculosis Sanitarium.

[1] Martin, C. L., and Sweany, H. C. Etiology of tuberculous anal abscess and fistula. Am. J. Digest. Dis. & Nutr. 1940, 7:36–39.

containing atypical giant cells, but about which there need be little question of a diagnosis.

In Figure 2 there is also chronic granulation tissue and a focal necrosis with a predominance of monocytic cells, some of which are becoming arranged into atypical giant cells of 4 to 8 nuclei. They are definitely not due to any false arrangement of cells of the rectal mucosa which may at times be confusing. The diseases of the rectum other than tuberculosis that may cause such a stimulation of the monocytic cells are few and largely hypothetical. It is granted that a tuberculoid condition may exist of the same nature of sarcoid lesions of the skin, although none has as yet been described. At the most, they would be too few to cause any distortion of the figures. The diagnosis, therefore, could safely be called a presumptive one until all the other data are correlated, or still better until staining of all the other sections and another search is made for more typical tissue. In our experience, when such a study is made, all but an insignificant minority fall definitely into one or the other group by the time one-half of the sections are examined.

SUMMARY AND CONCLUSIONS

The ultimate opinion concerning the nature of fistulous lesions of the anal region, therefore, will be influenced by various factors:

1. Clinical history and appearance of the lesion.

2. Clinical history of an active or quiescent tuberculosis or evidence of such by x-ray or laboratory examination.

3. The presence or absence of viable tubercle bacilli in the excised lesions demonstrated by culture or animal inoculation, but here the positive findings are subject to a 30 per cent error in positive sputum cases.

4. The most important and the only pathognomonic test when positive is the finding of tuberculous granulation tissue by histopathological study.

On a single section this is only positive in about 50 per cent of cases, but when run in serial section is at least 75 per cent efficient. Staining only every third section is usually all that is necessary, and even staining every tenth gives good results.

The absolute figures depend upon the number of the remaining 25 per cent that are non-tuberculous. If the animal or cultures are positive and the sputum is negative, such cases may be added to those found by histopathological examinations. If the specimens come from a positive tuberculous patient the probability is that it is tuberculous, but in the absence of the criteria above mentioned the lesions must be accepted as non-tuberculous or held in doubt until proved otherwise by the proper methods.

DYSGERMINOMA OF THE TESTICLE

A Clinical and Pathological Study of 30 Cases

JACK D. KIRSHBAUM, M.S., M.D., and MAURICE B. JACOBS, M.D., Chicago, Illinois

THE term "dysgerminoma" has been given to a group of tumors of the testicle which heretofore have been described as "seminoma" (Chevassu) "embryonal cell carcinoma" (Ewing); "germinal cell carcinoma" and "spermatocytoma" (Bell); and "round cell sarcoma" (McDonald).

In 1930, Robert Meyer, while investigating a group of testicular tumors, noticed their resemblance to the dysgerminoma of the ovary and because of this similarity he suggested the term "dysgerminoma" since he believed that these tumors originated from undifferentiated germ cells that are found in the gonads before sex differentiation occurs. Hinman and Ewing in this country and Bell in England are all in accord in the classification of the dysgerminomas as a type of a malignant teratoma in spite of the fact that only one type of cell may exist, as demonstrated in the dysgerminoma. The explanation offered is that due to the rapid growth of the type cell an overgrowth and suppression of the other elements occurs.

The tumors under discussion are malignant, often vary from the size of a walnut to that of a grapefruit, and histologically reveal large round or polyhedral cells with hyperchromatic oval to round nuclei and an ample cytoplasm. Hemorrhage and necrosis are frequently present.

In view of the diversity of opinion among various authorities as to the classification of this type of tumor, the term "dysgerminoma" as suggested by Robert Meyer may be an appropriate one.

As to the origin of these tumors, various authorities are fairly unanimous in their opinion that they are derived from the embryonic cells, while others such as Chevassu and Bell hold to the concept that the cells lining the seminiferous tubules give origin to these tumors. However, Robert Meyer believes that the cells are not derived from the tubules but rather invade them. Ewing is of the opinion that the tumor resembles or duplicates the epithelial cells of the spermatic tubules, i.e., the spermatocytes. Until the present time a suitable terminology has been found wanting, and therefore we believe the term "dysgerminoma" is descriptive of and applicable to this type of testicular neoplasm which in our study comprised over 95 per cent of all tumors of the testicle.

Our study consisted of a review of 30 malignant neoplasms of the testicle classified as "dysgerminoma." Of this group 12 patients came to autopsy (in a series of 12,000 consecutive necropsies from 1929 to 1939), and 18 patients were operated upon.

Twenty-seven of the patients were white and 3 were colored; as to age, 1 was between 10 and 20 years of age; 4 between 21 and 30;

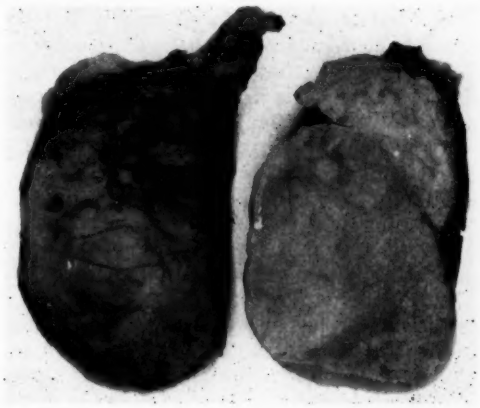

Fig. 1. Photograph of a dysgerminoma of the testicle. Left, Surface of tumor with intact capsule traversed by dilated and congested blood vessels. Right, section surface of the tumor. Note that the entire testicle has been transformed into a homogeneous firm tumor tissue.

From the Department of Pathology, Cook County Hospital, and the Division of Surgery, Northwestern University Medical School, Chicago.

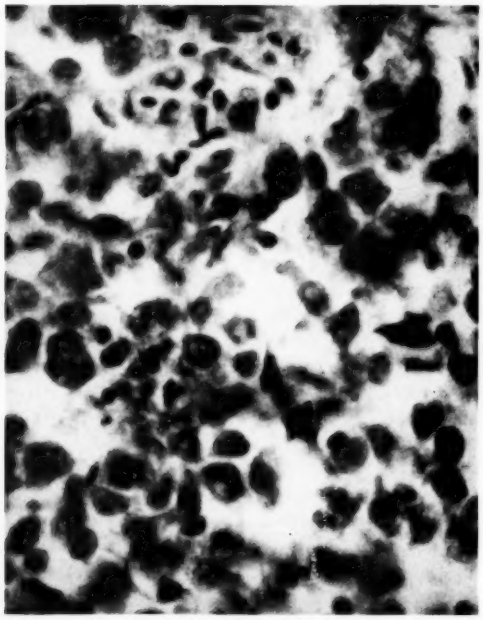

Fig. 2. Photomicrograph shows large and multinucleated cells, and a scanty stroma.

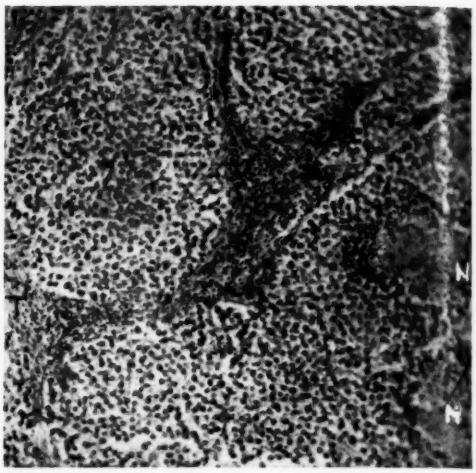

Fig. 3. Dysgerminoma of the diffuse type. Cells are interrupted by narrow strands of connective tissue, *T*, infiltrated with small round cells. Note 2 tubercle-like nodules, *n*, with central necrosis.

11 between 31 and 40; 6 between 41 and 50; 6 between 51 and 60; and 2 between 61 and 70. The third decade of life had the highest incidence, that is, 36.8 per cent.

The left and right sides were equally affected. Trauma to the testes preceding the onset of the tumor was admitted by 11 patients, or 36.8 per cent. The injury was in the form of either a blow, kick, or fall and occurred from 1 month to 15 years prior to the onset of a visible enlargement of the testicle.

Twenty-three or 76.6 per cent, of the patients presented swellings, which varied in size from a walnut to that of a grapefruit.

Loss of weight varied from 20 to 35 pounds in 10 of the patients, or 33.3 per cent, while 9 patients or 30 per cent, admitted suffering pain. The pain usually was described as moderate, while in the fatal cases lumbar pain was a frequent complaint.

As to the duration of symptoms in the 18 patients operated upon there were 10 between 2 and 6 months; 3 of 12 months; 3 of 24 months; 1 of 36 months, and 1 patient had noticed a swelling of the testicle for 6 years. In the 12 fatal cases, 7 came to operation and the survival period following the orchidectomy for the tumor varied from 1½ to 48 months.

An accurate clinical diagnosis of the tumor was made in 18 cases, or 60 per cent; the most frequent error in the diagnosis was hydrocele or tuberculosis.

SUMMARY OF THE PATHOLOGICAL FINDINGS IN 30 CASES

Grossly, the tumors varied in size from a walnut to a grapefruit (Fig. 1), were moderately firm in consistency, encapsulated, and on section were composed of a homogeneous grayish white tissue frequently mottled with purplish red areas of hemorrhage.

Histologically, the tumors revealed large polygonal cells often bordering one upon another with a scanty stroma. The cells were composed of an ample cytoplasm and contained large round to oval hyperchromatic nuclei. Nucleoli were often present and atypical mitotic figures were frequently seen. In some the nuclei were very large, irregular, and often multinucleated (Fig. 2).

In 20 of the patients evidences of hemorrhage were noted; in 25, necrosis. Five of the

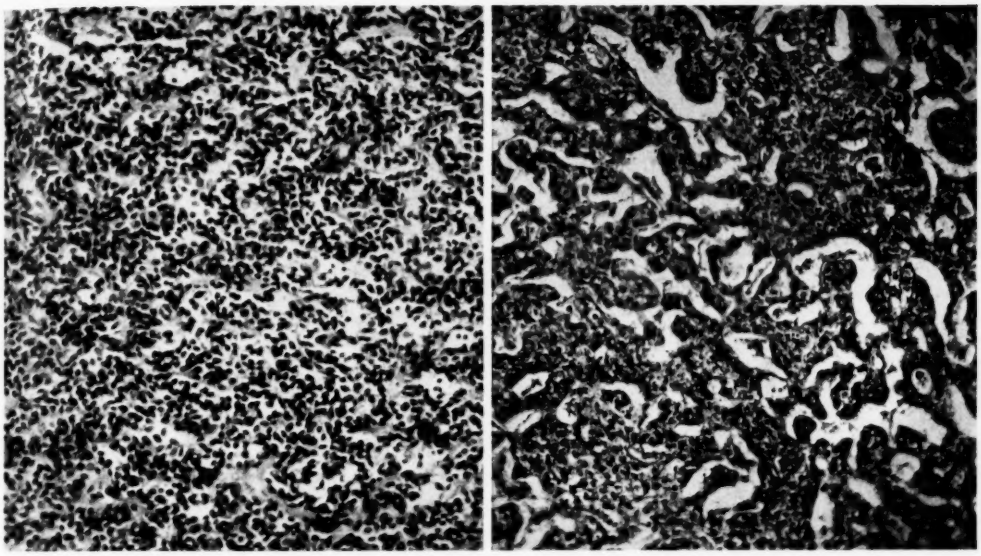

Fig. 4. Photomicrograph to show 2 types of the pseudopapillary form of dysgerminoma of the testicle.

latter revealed a caseation type of necrosis frequently forming circumscribed areas simulating tubercles. However, Langhans' giant cells were not observed (Fig. 3). In the fatal group of 12 patients no evidences of active tuberculosis were found at necropsy.

The cells comprising the tumor were arranged in a diffuse pattern in 20 patients, solid alveolar formation in 6, and a pseudopapillary type in 4. Of the latter 4 (Fig. 4), although the pseudopapillary arrangement of the cells was predominating, there were other areas in the same section which showed the diffuse type of arrangement of cells as was observed in the majority of the cases. The pseudopapillary type may be thus considered as a mixed form of a more highly differentiated type of dysgerminoma.

The stroma of the tumor was composed for the most part of narrow strands of connective tissue and accumulations of small lymphocytes, the latter varying in number, some of the tumors showing only scattered cells while in others the lymphocytes formed groups.

In the 12 cases that came to necropsy the opposite testicle revealed metastasis in only 1 instance, and in the remainder an interstitial fibrosis varied from a minimal to marked amount. The degree of spermatogenesis in the uninvolved testicle of the series varied, some showing normal activity while in others it was absent. The lymph nodes were involved in 10 cases and the distribution was as follows: peri-aortic, 10 times; mediastinal, 4; iliac, 3; peribronchial, 3; peripancreatic, 1.

Other sites involved in the metastasis were: lungs, 8 times; liver, 5; spleen, 1; right adrenal, 1; brain, 1; kidney, 1; pericardial sac, 1; diaphragm, 1; and spine, 1.

Thrombosis in the fatal cases was observed 5 times, or 41.6 per cent, and was found in the iliac veins and its tributaries.

NOMENCLATURE AND OTHER FEATURES

Although the term "dysgerminoma" for the ovary has been widely accepted both by pathologists and gynecologists, this has not been so with "dysgerminoma of the testicle." This may be explained by the fact that the terminology and the classification of tumors of the testicle is in quite a chaotic condition at the present time. It is not the purpose of this paper to propose a new nomenclature for testicular tumors, but up until the present

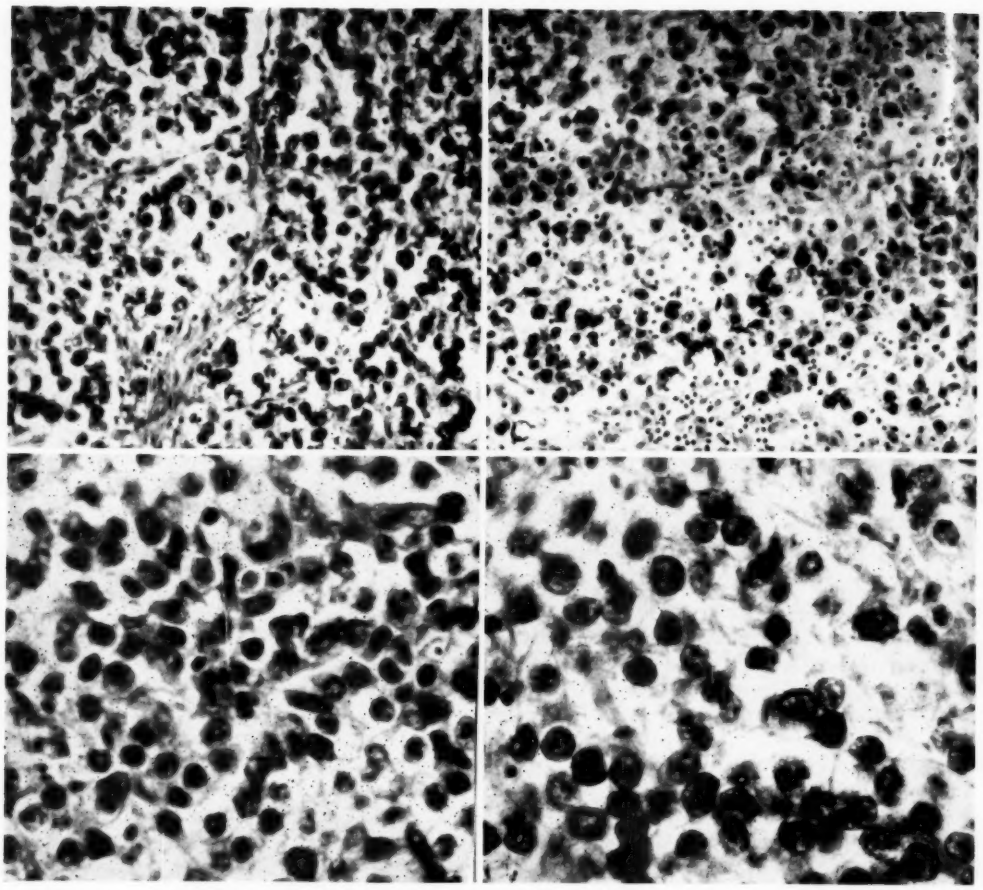

Fig. 5. Photomicrograph of dysgerminoma of the testicle, left, low power and high power. Note similarity to dysgerminoma of the ovary, right, low power and high power.

time a tumor of the testis has been called by any of 6 different terms. If tumors are to be classified on a morphological basis then the term "dysgerminoma" is fitting.

Although dysgerminoma of the testicle and the ovary simulate each other histologically (Fig. 5), clinically these 2 tumors differ in that the former is highly malignant and at necropsy metastases are extensive, whereas, dysgerminoma of the ovary frequently manifests itself by its benign course. However, in 53 reported cases of the latter, Doederlein found that 13, or about 25 per cent, had extrapelvic metastases and that 10 of these had run a fatal course. Novak states, "while dysgerminoma of the ovary is undoubtedly a malignant tumor, there are marked variations in the degree of malignancy of individual tumors. The outlook is very favorable when the tumor is unilateral with an intact capsule."

Non-descent of the testicle is often mentioned as a factor in predisposing to tumor development, but in our series not one of the tumors was associated with a previous non-descent of the testicle, although, in one case the opposite normal testicle was undescended.

SUMMARY

The results of a pathological study of 30 cases of malignant tumors of the testicle are given, twenty six of the cases were classified as dysgerminoma, while histologically 4 of the cases showed a mixed character. However, there were areas typical of dysgerminoma in the latter group and they may be considered as an intermediate stage of a more highly differentiated type of dysgerminoma.

The term "dysgerminoma" appears to be suitable in describing a distinct group of malignant tumors of the testicle or ovary, which heretofore have been known by a variety of terms, i.e., seminoma, spermatocytoma, embyronal cell carcinoma, etc., thus obviating much of the confusion that exists at the present time in the classification of testicular tumors.

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TREATMENT OF PATENT URACHUS

With Report of Seven Cases

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THE patent urachus is a condition which has been known to recorded medical literature since the time of Cabrol in 1550. No doubt patients with this affliction were seen prior to that time and the condition probably was recognized, but the cases were not recorded. This first case occurred in a female age 18 in whom there was obliteration of the urethral meatus associated with an umbilical tumor and fistula. The normal flow of urine was re-established, the umbilicus sutured, and the patient recovered.

Interest in the subject of patent urachus seemed to lag during the seventeenth century as no cases were reported. However, during the eighteenth century 4 cases came to light; 2 reported by Caulton and 1 each by Littré and Petit. Sixty-one cases are reported during the nineteenth century and 88 during the twentieth. This makes a total of 154 cases up to 1936 when Herbst (9) reported his and so ably reviewed the literature. Up to 1936 no one had reported more than 4 cases. Wilmoth and Hind each have reported 4.

In the literature there is seen a preponderance of males in a ratio of roughly 2 to 1. In my 7 cases there are 4 males and 3 females.

EMBRYOLOGY

Allantois and urachus are closely related terms and a clear understanding of their meaning and application must be gotten before the embryology and anatomy of the area can be understood.

The allantois is one of the first structures seen in the embryo. In his study of the development of the urachus in the human embryo, Begg follows its origin in common with that of the upper part of the bladder from the ventral cloaca. It must be borne in mind that the intra-abdominal portion of the allantois is called the urachus; while the allantois proper, including the urachus, runs from the top of the bladder out to its attachment to the placenta. The allantois is first seen as a saccule in the yolk sac. When the cloaca is formed, it (allantois) can be seen springing from that organ and running up through the umbilical cord toward the placenta. The bladder develops as a dilatation of the distal end of the allantois. At birth the allantois is normally present as a fibrinous cord in the umbilical cord. However, if the allantois remains patent, it is present as the patent urachus which may run as an open tube from umbilicus to the bladder or end as a blind pouch with no connection with the bladder. The abdominal part of the allantois, or to give it its true name, the urachus, is extraperitoneal in all of its course lying between peritoneum and the posterior sheath of the recti muscles.

TYPES OF URACHAL PATHOLOGY

1. *Extrophy of the bladder.* The bladder may open at the level of the umbilicus due to the high attachment of the bladder in the fetus, patent urachus, and deficiency of the anterior abdominal wall. These cases have, no doubt, often been classed as simple extrophy of the bladder.

2. *Congenitally patent urachus.* This is usually seen after the umbilicus sloughs off, but in some cases is seen on the day of birth. This may not develop until the child is several months old. This is often associated with stricture of the urethra and, at times, dilatation of the urethra to its normal size will cause symptoms to disappear completely. Prostatic hypertrophy in later life may cause symptoms in previously latent cases, but only after a marked degree of vesical neck obstruction has developed and been present for quite a time. Such cases are not uncommon.

3. *Patent urachus and omphalomesenteric duct in the same case.* There have been only

4 cases reported: Lexer 1898, Davey 1926, Davis and Niehaus in 1926, and Auslander and McClure 1930.

4. *Malignancy.* Cullen has reported 3 cases all of which had umbilical infection histories without much treatment. In 1921 Klopp reported an intramural urachal sarcoma which he treated by excision. There was complete recovery.

5. *Small urachal cysts.* These are of little importance and cause few symptoms.

6. *Large urachal cysts.* These often reach a very large size. Several have at one time or other been mistaken for and treated as pregnancies. Of course, time soon corrected this error. Rippmann reported one which contained 52 liters of fluid weighing 100 pounds. Most vary in size from that of an orange to a grapefruit.

7. *Abscess of the urachus.* This is the most common type of urachal pathology.

8. *Cavities between the umbilicus and bladder.* These are often seen. Such a case is my Case 6.

9. *Stones in the urachus.* These are rare. Four cases are reported: Paget in 1850, Dykes in 1910, Slovacek in 1929, and Smoler in 1932. All patients recovered.

10. *Tuberculosis of the urachus.* Tuberculosis is the rarest of all the complications. Only one case is reported. Eastman treated such a patient in 1915 by free dissection of the fistulous tract, which included the urachus, with recovery.

Of the 154 cases in the literature, the results are as follows: 107, or 69.6 per cent recovered; 13 or 8.4 per cent died; 28, or 18.1 per cent were not cured; in 6, or 3.9 per cent the result is not reported.

Of the 154 cases, 70 occurred in children under 13 years of age. Case 4 in my series was a child aged 1 year, with a large urachal abscess. This was my youngest patient. Rieder, De Geus, Mitchell, and Draudt each report a case occurring in the newborn.

The 3 most common types of urachal pathology are: large urachal cysts, abscess of the urachus, and cavities formed by the patent urachus between and connecting the bladder and umbilicus or with only one.

Urachal cysts. Large urachal cysts may vary from those containing a few ounces of fluid to the one reported containing 52 liters. These cysts are characterized by the gradual enlargement of the abdomen in the midline between the umbilicus and the pubic symphysis. The discomfort is usually due, if present, to tension on the abdominal wall. Physical examination shows a tumor mass in the suspected area.

Differential diagnosis must be made between distended bladder, ascites, appendiceal abscess, and ovarian cyst.

The cyst is treated by means of surgical removal of the cyst cavity down to the fundus of the bladder with inversion of the bladder stump as in inversion of an appendiceal stump. Sometimes it is necessary to remove the umbilicus by a circular incision.

Urachal cavities. One must consider whether the cavity opens only at the umbilicus and not into the bladder, whether it is connected only with the bladder and not with the umbilicus, thus forming an accessory or storage bladder, or whether it connects with both the bladder and the umbilicus. In Case 6 there is undoubtedly connection with both bladder and umbilicus because urine at times gushes from the umbilicus followed by disappearance of the tumor. At other times, the tumor will suddenly disappear following normal urination.

The symptoms are often those of the occasional case of pyelitis and the appearance and disappearance of the tumor mass between the navel and pubis. There may be an escape of urine in small amounts or a sudden gush from the umbilicus. This is often followed by the disappearance of the tumor for a while. Cystitis may develop in the accessory bladder as in the normal bladder.

Differential diagnosis must be made between distended bladder, ovarian cyst, and subcutaneous tumor or cyst.

If there is a great deal of infection present in the cavity, it is advisable to open and drain the sac. If possible, at the same time the bladder is separated from the sac and the vesical opening closed. The sac is then packed and allowed to contract down. If there is little danger of infection, the umbilicus is encircled and removed with the urachal sac and the bladder opening inverted.

Abscess of urachus. This condition is usually easy to diagnose if the umbilicus is examined,

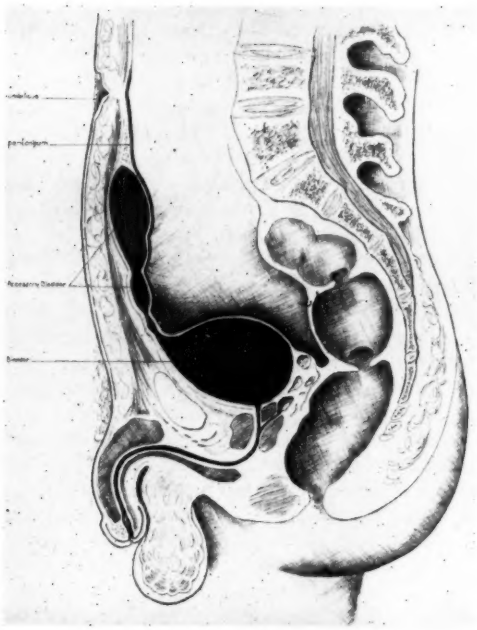

Fig. 1. Accessory bladder.

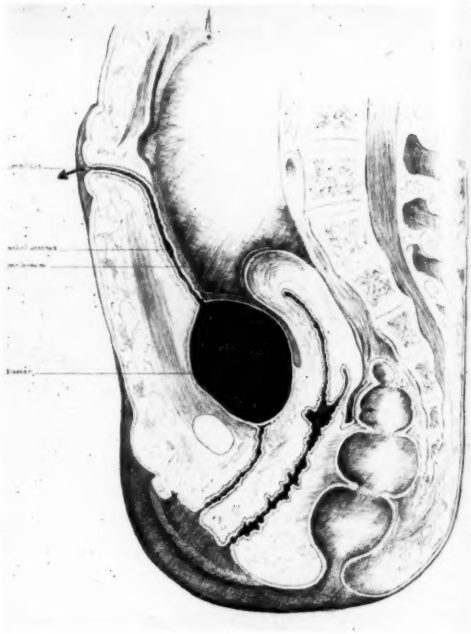

Fig. 2. Patent urachus.

as it is nearly always inflamed, discharging pus, and bulging. These abscesses are due to secondary infection of a urachal cyst or urachal cavity. The infection may be introduced through the umbilicus or ascend from the bladder following pyelitis. There is pain on pressure on the surrounding area with some tenderness in the lower abdomen. The other signs and symptoms of infection are present. Case 3 is an example of a large urachal abscess.

Differential diagnosis must be made between appendicitis, pelvic abscess, distended bladder, and retroperitoneal malignancy.

The treatment is incision and drainage with no attempt being made to remove the abscess sac as there is danger of opening the peritoneal cavity with resulting peritonitis. The important point to remember is that the patent urachus is extraperitoneal and that as long as there is only incision and drainage of the abscess no intra-abdominal damage will be done.

Following are brief histories of the 7 cases. Five can be listed as small cysts that had become infected and had to be removed. In 1, Case 3, there was a large urachal abscess. In Case 6 an accessory bladder had not come to operation as patient had been lost sight of. Of 7 cases, 6 patients were operated on with cure, while the other refused operation.

CASE REPORTS

Case 1. Mr. C. E. G. came in complaining of pain in the lower part of the abdomen, and drainage of pus from the umbilicus. At intervals, he had had a purulent discharge from the umbilicus all of his life. For the past 3 days the discharge had decreased and the pain increased. The past and family history was of no interest. Physical examination showed the abdomen flat and with tenderness in the lower portion. The umbilicus was swollen, red, and inflamed. No fluctuant mass was felt. A diagnosis of infected urachus with possible abscess was made.

At operation the urachus was found to be infected but without abscess formation. The urachus was patent down to but not into the bladder. The urachus was removed and the stump tied off and inverted. The abdominal cavity was accidentally opened. The patient recovered without complication.

Case 2. Mr. R. W. came in complaining of pain around the navel. About 2 weeks previously he had

begun to have pain around the navel, especially on the left side and down toward the bladder. There had been pus drainage from the umbilicus and at one time the patient "got the core out." The pain at one time went to the right side and for a while he thought that he had appendicitis: this later stopped. The pain was now present as a dull aching in the region of the navel. There was no past history of umbilical infection. There was a slight increase in the number of leucocytes, the temperature was 99 degrees F., and the pulse 84. All other physical signs were negative except in the umbilical region. It was inflamed and there was a purulent discharge. Slight pain and tenderness were present on the left and below the navel. At operation an infected urachus extending down about half way to the pubis was removed. The wound was closed with drainage and there was an uneventful recovery.

CASE 3. A baby girl, age 1 year, was brought in with a greatly swollen abdomen. The baby had been ill for the past 7 or 8 months. She had had repeated crops of boils that had not yet yielded to medical treatment. For the last 5 to 6 weeks the abdomen had been gradually swelling. There was no fever or apparent pain. There was a small amount of pus in the urine. The white blood count was 20,000; polymorphonuclears, 77 per cent; lymphocytes, 23 per cent.

On physical examination the child appeared to be acutely ill. There were many small pustules on the head and neck, and the color was poor. The abdomen showed a large, soft, dome-like mass extending from the pubis to just above the umbilicus, which was pushed up on top of the swelling. The swelling was dull on percussion, rather hard, and slightly tender on palpation. The umbilicus was distended to paper-like thinness. A diagnosis of abscess or cyst of the urachus was made.

At operation about a pint and a half of pus was drained from the abscess. The abscess cavity was packed with iodoform gauze and a hard rubber drain was left in the cavity. The cavity did not open into the bladder. The peritoneum was not opened. The child recovered rapidly and has had no return of symptoms or signs.

CASE 4. Mr. L. L. B. had a slight, irritating discharge from the umbilicus for 3 weeks, which had gotten worse. A week before presenting himself there was pain, swelling around, and discharge of pus from the umbilicus. He had had fever 101–102 degrees F., but no chills. At no time had there been a discharge of urine from the umbilicus. He had had several similar attacks in the past 15 years but none so severe as this. The leucocyte count was 12,000. The umbilicus was swollen and on pressure pus gushed from it. There was a swollen, inflamed area for about 10 centimeters around the umbilicus. Operation was advised.

At operation the urachus and umbilicus were cored out, a drain tube was put in, and the wound was closed. There was no connection between the bladder and the urachus. The peritoneum was not opened. The patient recovered without complication.

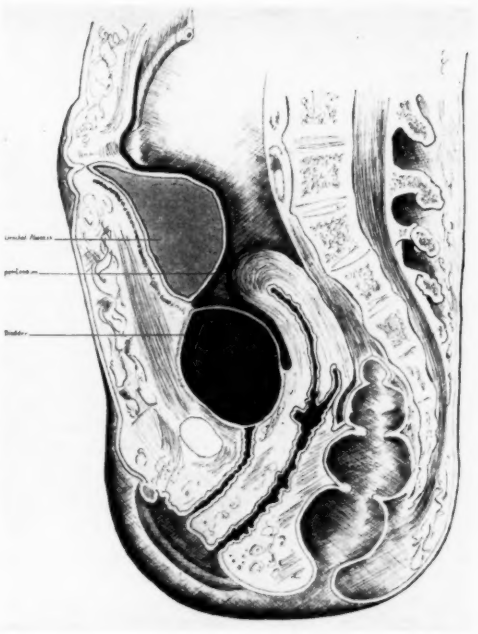

Fig. 3. Urachal abscess.

CASE 5. Mr. N. P. was seen in the office with an abscess pointing at the umbilicus. This was opened in the office. Local anesthesia was used. He was advised to come back in a month to have the urachus removed after the infection had died down. When seen 6 weeks later there was a slight discharge from the umbilicus; there had been no fever or chills and no active inflammation. The urachus was easily removed. There was no connection with the bladder.

CASE 6. A negro woman, aged 30 years, was seen in the City Clinic complaining of a small mass in her stomach. This mass on examination was found to be midway between the pubis and umbilicus and was in the midline. She stated that she had had this tumor for 10 years "off and on." At times there would be a gush of "water" from her navel and then the tumor would disappear or it might disappear following normal urination. At time of examination the tumor was present and pressure on it produced a few drops of urine at the umbilicus. I was unable to empty it by pressure, however. On cystoscopic examination, a small dimple was seen in the apex of the bladder. I could make out no frank opening, but I am sure that it was there because of the history.

A diagnosis of patent urachus with accessory bladder was made, and the patient was advised to have it removed as she complained of frequent attacks of severe cystitis. She refused and has been lost sight of.

CASE 7. Dr. W. Howard Wells has kindly allowed me to present this case. Mrs. R. C. F., aged 25 years, 6 months ago noticed a tenderness over the umbilicus. Two weeks later it began to drain. Her doctor burned it out with silver nitrate. A week later he burned it out with an electric needle. The silver nitrate was later used at weekly intervals. Because of lack of response and increased pain in the umbilical region, the patient came to the hospital for operation. On examination the umbilicus was found to be dark red and was exuding pus. There was tenderness on pressure over the umbilicus and down the midline toward the pubis. No masses were noted. A diagnosis of infected urachal cyst was made.

At operation an infected cyst 2 centimeters distal to the umbilicus was found and removed. There was no bladder connection. Recovery was prompt and she was dismissed from the hospital in 6 days.

SUMMARY

A review of the known literature on the patent urachus is presented with a report of 7 cases of patent urachus.

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THE PATHOGENESIS AND MECHANICAL PROPHYLAXIS OF VENOUS THROMBOSIS

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THE problem of thrombo-embolism occupies a central position in medical study. During the course of past years, innumerable investigations have been carried out regarding it but without much deeper penetration into its pathogenesis having been accomplished. The reason for this seems to be that the pathologico-anatomical side of the question has previously been incompletely studied.

According to earlier opinion, which has been generally accepted since the time of Virchow and which has been especially maintained by Aschoff and his school, the femoral vein and its valve-pockets are the most frequent points of origin of deep-lying venous thrombi of the lower extremities. It has been assumed that, as a result of the agglutination of thrombocytes and white blood corpuscles, a white thrombus is formed which gradually obstructs the vessel. Then, it is said, a red "coagulation-thrombus" arises in the mass of blood which is dammed up, peripheral to the primary thrombus.

This hypothesis agrees with the clinical observation that patients suffering from thrombosis feel tenderness over the femoral vein and eventually, and simultaneously with increased pulse and heightened temperature, display edema and cyanosis of the lower leg.

However, Denecke, in 1929, and Olow, in 1930, proved that the earliest clinical symptoms of thrombosis appear in the sole of the foot and in the calf. The assumption which almost immediately presents itself is that these symptoms are an expression of the very earliest formation of the thrombus, and that the process then continues upward together with the blood stream, and not, as was formerly supposed, in a retrograde direction from the femoral vein. Homans, in 1934, supported by clinical and pathologico-anatomical observations, emphasized the great importance of the veins of the calf as areas of origin of an ascending thrombus.

It was first in 1937–1938, however, that Roessle and Neumann published the results of an extensive series of pathologico-anatomical investigations, in which there was fully demonstrated the dominant rôle played by the plantar veins and the veins of the calf as the seats of origin of thrombosis.

The year before the work of the first of these authors was published, the present writer, then amanuensis at the pathological department of the Caroline Institute, Stockholm, of which Professor F. Henschen was chief, commenced work on a series of problems to determine the commonest areas of origin of deep-lying venous thrombi. A detailed report of his results was published (4).

The frequency of thrombosis as found in different vein sections in my series is shown in Figure 1. The greatest incidence is in the peripheral veins of the calf and adductor musculature, and grows less toward the center.

The average age of the patients in my series is very great, consequently plantar venous thrombi are very sparingly represented. Neumann has demonstrated that such thrombi occur usually in young persons, while, with increasing age, the veins of the calf become more and more the chosen seat of an incipient thrombosis. Obstetrical and gynecological instances are also rare, and this is probably the reason pelvic thrombi are relatively few in number.

Earlier theories regarding thrombosis have taken into consideration a process of retrograde thrombosing from the larger calibered vessels back into the peripheral veins. In my series, however, the anterior tibial vein has never been thrombosed in spite of the fact that the site of entrance into the popliteal vein has been blocked by thrombi in no less

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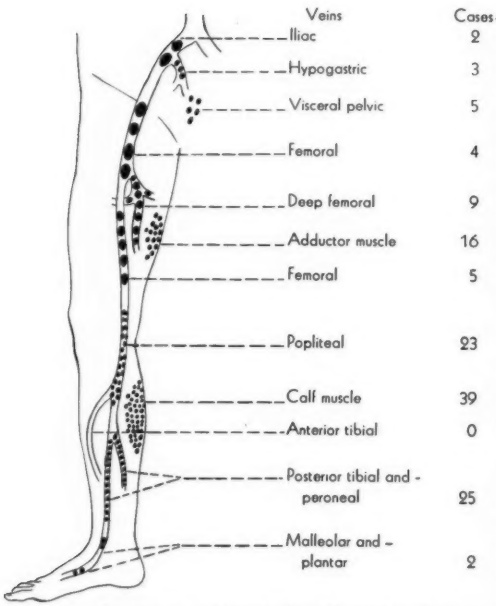

Fig. 1. Sites of venous thrombosis with number of cases for different veins of lower leg.

than 23 instances. Further, I have observed cases in which the adductor veins, the deep femoral vein, and the upper part of the femoral vein have been thrombosed, while the lower part of this latter vein has remained free. Finally, it is demonstrated in my series of cases that the veins of the calf and all of the femoral vein have been affected with thrombosis, while the branches of the deep femoral vein have remained free from thrombi. It is therefore questionable whether a retrograde thrombosis ever occurs against the direction of the blood stream.

If my series of investigations are compared with those of Roessle and Neumann, we find that there are four areas of origin of incipient thrombosis, namely: (1) the plantar veins, (2) the veins of the musculature of the calf, (3) the veins of the adductor musculature, and (4) the visceral pelvic veins. From these areas of origin, the thrombosing process follows the blood stream and grows into the large calibered veins.

It has long been recognized that the pelvic veins are important as points of origin of thrombosis. That the plantar veins and the veins of the calf play a similar rôle has been demonstrated, especially in the investigations of Roessle and Neumann. On the other hand, the importance of the veins of the adductor region as an area of origin of an ascending thrombus has not previously been sufficiently studied. I believe, therefore, that it is probable in cases in which earlier investigators have observed thrombosis of the femoral vein but none of the calf, that the process perhaps has usually started in the deep femoral vein and was not a primary femoral thrombus.

More recent, wider knowledge of pathologico-anatomical conditions enables us to assume a number of new points of view regarding the problem of thrombo-embolism.

First, we may make the deduction that the four areas of origin mentioned are influenced by some special local factors connected with the patient's position in bed and that these factors gradually lead to injury of the intima of the vein. As a rule three general conditions for the rise of a thrombus must be taken into consideration, namely: (1) a slowed-down blood stream; (2) changed chemical composition of the circulating blood; (3) injury to the intima. The first two factors are merely contributory ones in the pathogenesis and alone can never give rise to a thrombus. We know that it is possible to ligate a vein without a thrombus being formed under the presupposition, of course, that the intima is not injured in the process (Klemensiewicz).

The composition of the blood is changed, of course, in all morbid conditions and also after operations which apparently are aseptic. The alterations consist, *inter alia*, of the globulin-concentration of the blood-plasma undergoing a relative increase in proportion to the albumin concentration, whereby the colloidal equilibrium of the blood-plasma becomes less stabile (Hueck, Loehr, Starlinger, Heusser, and others). The blood thus acquires an increased tendency to coagulate but, in this connection, it is to be observed that the entire vascular system is exposed in equal measure to an increased risk of becoming thrombosed. As a matter of fact, numerous examples have been brought forward which show that venesection is never entirely without this danger. Conse-

quently, the changed chemical composition of the blood can by no means explains why a thrombus so often arises just within the four areas of origin mentioned.

On the other hand, a sufficiently severe injury to the intima, even without the association of other factors, can always give rise to a thrombosing process. This has been verified by numerous experiments with animals, and is an everyday experience in operations. The technique used in the experiments, however, has been non-physiological (cauterizations, and the like), so that it has not been possible to accept fully the results of the experiments as an explanation of the origin of a spontaneous thrombosis in man.

What are the factors regarding the circulation in the lower extremities of a patient confined to bed? Roessle has directed our attention to the fact that in such positions, the calves lie pressed flat against the mattress. As a matter of fact, the adductor region likewise is exposed to similar mechanical pressure, partly from below and partly from the median side; this is especially true in fat individuals whose thighs are usually pressed tight against each other.

Moreover, I wish to point out that it is just these two groups of muscles which are least used for active muscular movements by patients in bed. Movements then are usually restricted to the occasional bending of the knees upward, a movement which is carried out chiefly by contraction of the psoas and rectus femoris muscles. Then follows the straightening of the legs by means of their own weight, with some slight assistance from the quadriceps muscle of the thigh and the great gluteal muscle.

When a patient is confined to bed, the flow of arterial blood to the extremities diminishes in consequence of the lessened need for such blood. The total quantity of blood which, per time-unit, passes through the extremities is therefore diminished, and the comparatively greatest diminution must, in consequence of what has just been said, affect only the musculature of the calves and the adductor muscles.

As we know, in the venous system of the extremities varicose alterations are extremely common. The veins of the calves, especially,

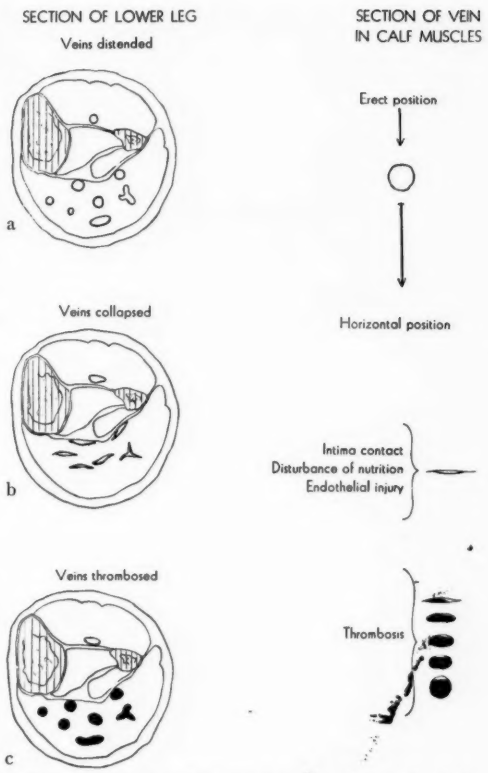

Fig. 2. Sections of the lower leg, left; section of vein in calf muscles.

have extremely thin walls, and they are especially wide as compared with the corresponding arteries and lie directly embedded in a heavy mass of muscle.

In an erect position, the veins are distended to the maximum with blood, as upon them lies the pressure of a considerable venous blood column (Fig. 2a).

In a lying position, on the other hand, the pressure on the veins diminishes; the veins therefore collapse more or less completely. This is easily observed in superficial varicose veins. Especially marked should be the collapse of the veins in the calf and adductor regions, which are exposed to mechanical pressure (Fig. 2b).

One may then very well take into consideration the fact that, to a very great extent, in-

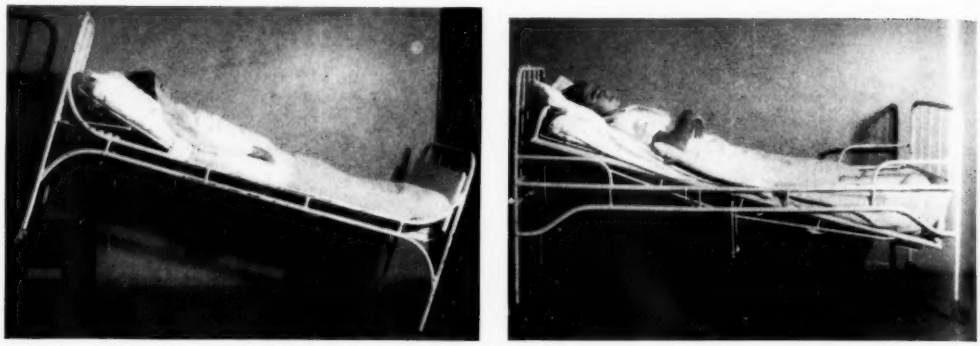

Fig. 3. Elevated head-end, to diminish the tendency to thrombosis.

tima will lie pressed against intima, while a minimal stream of blood will percolate onward through a crack-like lumen which remains open in some part of the vessel.

However, there is reason to suppose that the vitality of the endothelial cells depends in high degree on their contact with flowing blood, to which the products of metabolism are delivered and from which, eventually, nutrition is obtained. When the endothelial cells are deprived of this source of nutrition, disturbances arise in them. Perhaps a purely mechanical adhesion of the endothelial layers occurs, so that some of the cells become defective or are torn loose when, later on, the vessel becomes distended. However it may be, there is every reason to suppose that, a somewhat prolonged contact of two intimal layers gradually produces an injury to the endothelium, thus bringing about a fundamental condition for the formation of a small thrombus.

From the surface of this thrombus, however, substances are given off to the passing blood and these promote coagulation, so that layer after layer of the blood-mass becomes coagulated. The process moves onward together with the circulating blood in a longitudinal direction, until finally, a vein which before was pressed together becomes completely distended with the thrombotic masses (Fig. 2) and regains its circular contour. Figure 2c shows a section of the calf after complete thrombosis.

As to the finer structure of a thrombus, the rule probably holds good that a white thrombus arises in an area where the stream speed is great. Then the concentration of the substances which cause coagulation rapidly diminishes, and it will be mainly the leucocytes which will stick fast to the surface giving off the substances in question. (As we know, the leucocytes follow the walls of the vessels while the red blood corpuscles are arranged chiefly in the center of the blood stream.) The trabecula-shaped structure of the white thrombus is explained as arising from "whirl"-formations, as shown by Aschoff in his experiments.

If, on the other hand, the speed of the blood stream is slow, a red thrombus will arise, as the substances causing coagulation will have the opportunity to accumulate a larger mass of blood, in which process all the formative elements of the blood will be enclosed in a network of coagulated fibrin.

That is the reason we usually find a white thrombus in the upper part of the femoral vein, at the place where the deep femoral vein opens into this vessel. For, whether the process originates in the calf or in the adductor region, the uppermost end of the onward-growing thrombus mass will, at the place aforementioned, be washed past by a rapid blood-stream, and thus give rise to a white thrombus. It is this latter which, in Aschoff's thrombus-theory, is called the "thrombus-head" understood as being the primary part of the thrombus mass. Today, however, such an hypothesis could probably hardly be maintained.

In the reasoning given, I have had in mind the veins of the calf and of the adductor mus-

Fig. 4. a, Palpation of the calf and adductor regions, for making an early thrombus diagnosis. b, Palpation of thigh.

culature, but the same arguments can be used to some extent in respect to the two other sources of origin of thrombi, namely, the plantar veins and the pelvic veins.

For, under normal conditions, the plantar veins are kept distended by the highest blood column existing in the human body. One can then certainly take for granted that, in many persons who are confined to bed, these veins will usually lie collapsed.

As regards the rise of thrombi in the pelvic veins, probably an important rôle is played in the matter by infections, toxic products, and traumatism during operation. But in the pelvis, too, one may certainly suppose that altered meteorism, pregnancy, etc., possibly in conjunction with a failing circulation, may cause the veins to be insufficiently distended and, in consequence, become the seat of a thrombus, in analogy with the process described in the case of the extremities.

PROPHYLACTIC MEASURES

If these ideas regarding pathogenesis are correct, then the prophylactic measures hitherto employed must be altered. Formerly all mechanical measures have aimed at the emptying of the venous system of the extremities and to do this the foot of the bed has been raised or diverse other mechanical measures have been used. Such measures, however, help to aggravate conditions which must be present if collapse of the veins and adhesions of endothelial cells are to occur. During the past few years the results of prophylactic measures used have been so unsatisfactory—in some instances the increase in number of cases of thrombosis has been as much as tenfold—that there is every reason, supported by the theory here presented, to try new methods of prophylaxis.

During the past year at the surgical department of the Karlshamn County Hospital, I have had the opportunity of applying another method in a number of surgical and delivery cases which had been considered especially hazardous in regard to thrombosis. The patients had formerly had thrombi, or presented very unpromising varicose legs. Judging from all appearances, they had typical thrombus constitutions.

The method used was as follows: For 1 to 2 hours daily, the head of the patient's bed was elevated by placing the two head-legs upon chairs (Fig. 3). The patient then began to slide downward in the bed, and had to press her feet against the foot of the bed in order to maintain her position. She automatically made slight movements with the knees and changed the position of the feet every now and then, thereby lessening the pressure on the musculature of the calves and adductors. In the movements thus carried out, it was necessary to use both these groups of muscles to make active contractions. The venous pressure in the extremities rose, and this helped to maintain the distention of the veins.

The position causes but very slight unpleasantness to the patient, and none of them thus treated had any thrombi. During the same period, however, we had several cases of throm-

bus among patients not treated as described. One died of a pulmonary embolus.

It is true that at times a condition of collapse after operation makes it necessary for the patient to lie with the head low, but if care be taken to keep the venous system distended with blood for an hour or so each day, I believe that the tendency to thrombus formation will be counteracted to a high degree even if not entirely so.

As a further argument for this treatment, I should like to point out that, as a rule, a patient remains free from thrombi if it is possible to allow her to be up after a few days following operation. If this is not possible, an endeavor should be made to place the patient in a position which resembles the upright as far as possible, and *not* in a position with the legs high.

The results in the treatment of thromboembolism depend, however, to a very great extent on the ability to make an early diagnosis. Diagnosis must be made long before the signs of a more massive thrombosing become evident in the form of cyanosis, edema, and increased pulse and temperature. Before a patient is allowed to leave her bed, therefore, extremely careful palpation must be made of the four areas of source from which a thrombus usually comes. Palpation of the calf (according to Olow) should be carried out as shown in Figure 4, a, with both knees symmetrically semiflexed, the musculature of the extremities thus becoming completely relaxed. It is then possible to palpate the calf-musculature exactly on both sides. Then, the medial-posterior part of the thigh is thoroughly palpated bimanually, and with gentle movements of the hands (Fig. 4, b). I have found that a slight increase in consistency, or tenderness in deep-lying parts within this area, is a valuable early symptom of thrombosis.

SUMMARY

1. Several series of pathologico-anatomical investigations have been made during past years and have proved that there are four areas of origin of venous thrombosis: (a) the plantar veins, (b) the veins of the musculature of the calf, (c) the branches of the deep femoral vein in the adductor musculature, and (d) the visceral pelvic veins.

2. When a patient is confined to bed, the veins of the areas mentioned, to a certain degree, are collapsed, or pressed together, so that two intima layers come into close contact.

3. The vitality of the endothelial cells depends, to a great extent, on their contact with flowing blood, and when the cells are deprived of this source of nutrition, disturbances arise in nutrition, and a thrombosing process is begun.

4. Injury to the intima is the most important factor in the pathogenesis of thrombosis. It can be counteracted by raising the head of the bed so that the patient begins to slide downward in bed. Then the venous pressure in the extremities will rise, so that the veins become distended with blood, and the patient will be forced to make active movements with her legs to maintain her position. Thus the veins which are especially threatened by thrombosis will be rhythmically emptied and distended.

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VITAMIN B_1 DEFICIENCY IN PREGNANCY AS INDICATED BY A TEST FOR OBT PRINCIPLE

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NUMEROUS articles have appeared indicating that a deficiency of vitamin B_1 occurs frequently in pregnancy and that this deficiency may be overcome by administration of the vitamin. The symptoms of this deficiency have been reported as polyneuritis, hyperemesis gravidarum, numbness and tingling of the fingers and toes, tenderness of the extremities to pressure, muscular weakness, loss of deep reflexes, anorexia, periodic nausea, glossitis, tachycardia, edema, easily induced fatigue, etc.

One of us has reported a quantitative biological test of blood serum for estimating the effect of vitamin B_1 administration. The substance appearing in the blood, the level of which seems to be an index of the activity of vitamin B_1, has been given the name "OBT principle." The details of the technique of the test for "OBT principle" will be found in a previous article (4).

EXPERIMENTS

One hundred pregnant women were studied. Determinations of OBT titer was made on each at some time during the last trimester of pregnancy. After this preliminary titration for "OBT principle," each was given yeast with instructions to eat 3 cakes a day over a 4 day period and then to return for another determination of OBT titer.

It has been determined by one of us that the substance in yeast responsible for the rise in OBT titration of the serum is vitamin B_1 (thiamin). Yeast was used because of its low price as compared with that of thiamin (5). There were 300 international units of vitamin B_1 in each yeast cake.

ANALYSIS

Analysis is made (see chart) of these patients as to age, race, development of neuritis, development of palpitation, type of delivery, months pregnant, number of pregnancies, development of toxemia (including hyperemesis gravidarum), weight at beginning and just before termination of pregnancy. Due to insufficient data only 86 of the 100 patients were included in the analysis. However, we are fortunate in being able to include several patients who had a low serum titer for OBT. Each patient was interviewed personally by one of us in order to evaluate more accurately information which was given by each of the patients.

Thirteen patients were found to be low in vitamin B_1. Their average age was 26½ years, 9 of the 13 were white, the rest negresses. Ten of the 13 developed neuritis. One of the patients was delivered by cesarean section, the rest vaginally. All 13 were full term pregnancies. Seven of the 13 had hyperemesis gravidarum early in their pregnancy. Six of the 13 had definitely poor appetites. Their average weight in the first trimester was 133 pounds, just before delivery the average weight was 156 pounds.

Of the 73 patients with a normal or high titer, 57 were white, the rest negresses. Their average age was 25 years. Nine of the 57 developed neuritic symptoms—of these 7 had a low normal titer, 2000. Twenty-four had hyperemesis gravidarum early in pregnancy—OBT titer in these patients varied widely. One of these patients developed postpartum eclampsia and died. OBT titer in this patient during pregnancy was 5,000 before and after ingestion of yeast. It was repeated during eclampsia seizure and remained at 5,000. Only 10 patients had a poor appetite and 8 of the 10 had a normal low titer of 2,000. Their average weight in the first trimester was 130 pounds, just before delivery 152 pounds. In 77 of the 79 patients who returned after ingesting 12 yeast cakes, the OBT titer was definitely raised. The rise was assumed to have resulted from vitamin B_1 (5).

From Medical Service A, Pennsylvania Hospital.

ANALYSIS OF CASES

No.	Initials	OBT titer Before yeast	OBT titer 4 days after yeast	Age	Race	Neuritis	Palpitation	Edema	Type of delivery	Mos. pregnant	Para	Toxemia	Appetite	Weight before pregnancy	Weight after delivery
1	J. S.	1,600	5,000	27	w	+	+	–	OF	9	1	sl h	good	151	171
2	M. J.	1,600	Did not return	31	c	–	occ	–	S	9	7	sl h	fair	128	142
3	C. P.	1,600	Did not return	22	w	+	occ	–	S	9	2	h	fair	95	120
4	A. F.	1,600	5,000	26	w	sl	–	++	LF	10	1	h	fair	166	190
5	J. S.	1,600	5,000	30	c	++	–	+	LF	9	1	h	poor	88	114
6	J. F.	1,600	2,500	23	w	++	+	+	LF	9	1	h	O. K.	155	185
7	A. B.	1,000	5,000	23	w	++	+	+	S	9	4	h	O. K.	123	140
8	I. H.	1,600	5,000	16	w	+	–	+	OF	8	1	–	O. K.	105	119
9	M. M.	1,600	5,000	24	w	sl	–	–	LF	9	1	h	O. K.	111	136
10	L. D.	1,250	Did not return	29	w	+	–	++	LF	9	1	–	O. K.	195	211A
11	E. W.	1,450	5,000	18	c	++	+	+	CS	8½	2	–	fair	136	160
12	Mrs. K.	1,600	5,000	20	w	sl	–	++	S	8	1	–	poor	135	171
13	Mrs. V.	1,600	5,000	36	w	+	–	+++	S	9½	7	h	fair	?	172
14	A. H.	2,500	3,300 (ate 6 cakes)	28	c	–	–	–	LF	9	1	–	good	163	190
15	A. J.	2,000	10,000	31	w	sl	–	sl	OF	9	1	–	good	141	167
16	M. B.	2,000	5,000	30	c	+	–	–	S	9	2	–	good	165	177
17	M. M.	2,000	5,000	24	w	–	–	–	S	9	4	–	good	?	122
18	A. M.	3,300	10,000	28	w	–	–	–	S	9	1	–	good	133	151
19	E. C.	5,000	5,000	25	w	–	–	–	S	9	1	–	good	?	?
20	S. M.	5,000	5,000	23	c	+	sl	sl	S	8	3	B	good	98	126B
21	H. M.	3300	10,000	21	w	–	–	–	S	9	1	–	good	155	165
22	S. W.	3,300	5,000	29	c	–	–		OF	9	1	–	good	136	139
23	R. P.	2,000	Did not return	25	w	sl	–	–	LF	9	1	–	good	113	133
24	I. S.	2,500	5,000	24	c	–	–	–	S	9	2	–	good	110	133
25	A. C.	3,300	10,000	24	c	–	–	–	LF	9	2	–	good	133	160
26	M. B.	2,500	12,500	19	w	sl	–	sl	OF	9	1	h	good	111	133
27	A. L.	3,300	10,000	33	w	–	–	–	S	9	r	h	good	144	165
28	C. R.	2,000	3,300	23	w	sl	–	sl	S	9	2	–	good	172	202
29	M. S.	2,000	10,000	31	w	–	sl	sl	S	9	6	–	good	182	198
30	A. Y.	5,000	10,000	22	w	–	–	–	S	9	2	h	good	158	175
31	D. S.	3,300	10,000	26	w	–	–	–	S	9	3	–	good	133	145
32	J. F.	3,300	Did not return	23	w	–	–	–	LF	9	2	h	good	132	151
33	M. VS.	2,000	10,000	23	w	+	–	–	S	6½	2	–	good	107	122C
34	A. P.	2,000	10,000	30	w	–	–	–	S	9	3	–	good	162	178
35	N. R.	2,500	14,500	32	w	–	–	–	LF	9	2	–	good	106	129
36	C. D.	2,500	8,000	27	w	–	–	–	LF	9	1	–	good	118	140
37	L. F.	2,500	5,000	26	w	sl	–	+	LF	9	1	–	good	120	152
38	M. S.	3,300	10,000	27	w	–	–	sl	S	9	2	–	good	136	170
39	P. D.	3,300	10,000	29	w	–	+	+	LF	9	1	–	good	130	155D
40	I. C.	2,000	5,000	33	w	–	–	–	MF	9	2	–	good	205	214
41	N. T.	2,500	5,000	21	w	sl	–	++	MF	9	1	–	good	126	161
42	P. B.	2,000	11,500	29	w	–	–	–	S	8	2	–	fair	133	148

ANALYSIS OF CASES, *Continued*

No.	Initials	OBT titer Before yeast	OBT titer 4 days after yeast	Age	Race	Neuritis	Palpitation	Edema	Type of delivery	Mos. pregnant	Para	Toxemia	Appetite	Weight before pregnancy	Weight after delivery
43	R. M.	2,000	5,000	18	w	+	−	+	LF	9	1	sl h	good	122	152
44	N. T.	2,000	5,000	31	w	−	−	+	S	9	2	−	good	139	153
45	M. L.	2,000	10,000	28	w	−	−	−	S	9	2	−	good	106	125
46	C. G.	2,500	10,000	19	w	−	−	−	S	9½	2	−	good	135	168
47	C. L.	3,300	10,000	23	w	−	−	−	S	9	1	h	good	144	177
48	C. D.	3,300	8,000	33	w	−	−	−	S	9	3	−	good	118	136
49	M. C.	2,500	5,000	22	w	−	−	−	S	9	2	−	good	104	124
50	H. A.	3,300	5,000	28	c	−	−	sl	S	9	4	−	good	163	190
51	G. D.	2,000	5,000	28	w	−	−	+	S	9	2	h	good	107	129
52	C. R.	2,500	3,300	19	w	−	−	−	OF	9	1	h	good	95	130
53	B. N.	2,000	5,000	26	w	sl	−	−	LF	9	1	h	good	122	155
54	K. B.	2,500	10,000	23	w	−	−	sl	LF	9	2	−	good	139	164
55	K. R.	2,000	20,000	18	c	−	−	sl	OF	9	1	−	good	111	131
56	A. D.	2,000	3,300	28	w	sl	+	++	S	9	1	h	good	130	166
57	J. C.	2,500	Did not return	24	w	−	−	sl	LF	9	1	−	good	?	154
58	C. G.	3,300	Did not return	18	w	−	−	−	OF	8	1	−	good	113	140
59	E. M. J.	2,000	10,000	27	w	−	−	sl	Br	9	2	−	good	202	218
60	A. M.	2,000	3,300	27	w	−	−	sl	CS	9	1	−	good	101	120
61	S. B.	2,500	Did not return	21	w	−	−	+	LF	9	1	−	good	121	147
62	T. A.	2,500	Did not return	27	w	+	−	+	S	9	1	h	good	150	180
63	F. W.	2,000	5,000	28	w	+	−	+	Br	9½	1	−	fair	131	161
64	M. B.	2,500	5,000	25	w	+	−	++	S	9	3	−	good	179	223
65	S. M.	2,000	10,000	22	w	+	−	+	OF	9	1	h	good	?	157E
66	M. T.	500	10,000	22	w	+	−	++	OF	9	1	−	poor	116	155
67	A. S.	2,000	10,000	20	c	+	−	−	S	9	2	−	good	134	152
68	A. D.	3,300	Did not return	27	w	−	−	sl	CS	9	2	−	good	118	128
69	E. D.	5,000	Did not return	28	w	−	−	−	S	9½	2	−	good	150	175
70	K. C.	2,500	3,300	14	c	−	−	−	LF	8½	1	h	good	98	114
71	G. P.	2,500	Returned (Did not eat yeast)	21	w	−	−	−	CS	8	2	−	good	96	122
72	J. B.	2,000	10,000	30	w	+	−	++	S	9	11	h	good	129	146
73	K. S.	10,000	Did not return	24	w	−	−	+	LF	9	2	h	good	130	164
74	Mrs. S.	3,300	10,000	18	c	−	−	−	MF	9	1	−	good	110	130F
75	M. J.	2,000	10,000	20	c	−	−	−	LF	9	1	−	good	?	136
76	Mrs. C.	3,300	10,000	22	w	−	−	+	LF	9	1	−	good	95	123
77	Mrs. F.	2,000	Did not return	22	w	−	−	++	LF	9	1	−	good	122	158
78	Mrs. S.	2,000	5,000	17	c	−	−	−	LF	9	1	−	good	114	143
79	J. F.	2,500	4,000	22	c	−	−	−	S	9	2	h	good	84	107
80	M. M.	3,300	Did not return	21	w	−	−	+	LF	9	1	h	good	133	161

ANALYSIS OF CASES, *Concluded*

No.	Initials	OBT titer Before yeast	OBT titer 4 days after yeast	Age	Race	Neuritis	Palpitation	Edema	Type of delivery	Mos. pregnant	Para	Toxemia	Appetite	Weight before pregnancy	Weight after delivery
81	Mrs. C.	3,300	Did not return	23	w	—	—	+	MF	10	1	—	good	136	155
82	M. Z.	3,300	5,000	36	w	—	—	—	S	9	3	—	good	153	172
83	Mrs. B.	3,300	Did not return	19	w	—	—	—	S	9	3	h	good	88	104
84	J. G.	3,300	10,000	19	w	—	—	—	LF	9	1	h	fair	146	157
85	Mrs. B.	2,000	10,000	33	c	—	—	—	S	9	2	—	good	?	176
86	Mrs. C.	2,500	5,000	25	w	—	—	—	S	10	3	h	fair	118	123

w—white c—colored S—spontaneous Br—breech
OF—outlet LF—low forceps MF—mid forceps
h—hyperemesis gravidarum sl h—slight hyperemesis gravidarum
—negative +—definitely present ++—very definite
A—Blood pressure 150/100 at start of pregnancy; later fell to 120/80.
B—Postpartum eclampsia resulting in death of mother and child.
C—Compensated rheumatic heart disease.
D—Premature baby.
E—One month after test, thiamin chloride was given, 300 units, t.i.d., for one week for cramps in lower legs with beneficial results 10 days after the start of thiamin.
F—Treated as obesity preventative, edema of hands and feet and polyhydramnios.
occ—Occasional

SUMMARY AND CONCLUSIONS

1. Thirteen pregnant women, from a total of 100, studied by a test on blood serum for the effect of vitamin B_1, were found to be low in titer. This would indicate the frequency of this deficiency in pregnancy.

2. The OBT level—details of test given in previous article—can be raised by the administration of yeast over a 4 day period.

3. Patients judged to be low in vitamin B_1 by this test have a definite tendency to develop neuritic symptoms.

4. One patient who developed eclampsia, had an adequate titer of OBT.

5. From this study it would appear that a deficiency of vitamin B_1 in pregnancy has a definite effect upon the appetite.

We desire to thank Dr. Norris Vaux, Dr. Robert A. Kimbrough and Dr. Clifford B. Lull for permission to use the listed cases of pregnancy from their services.

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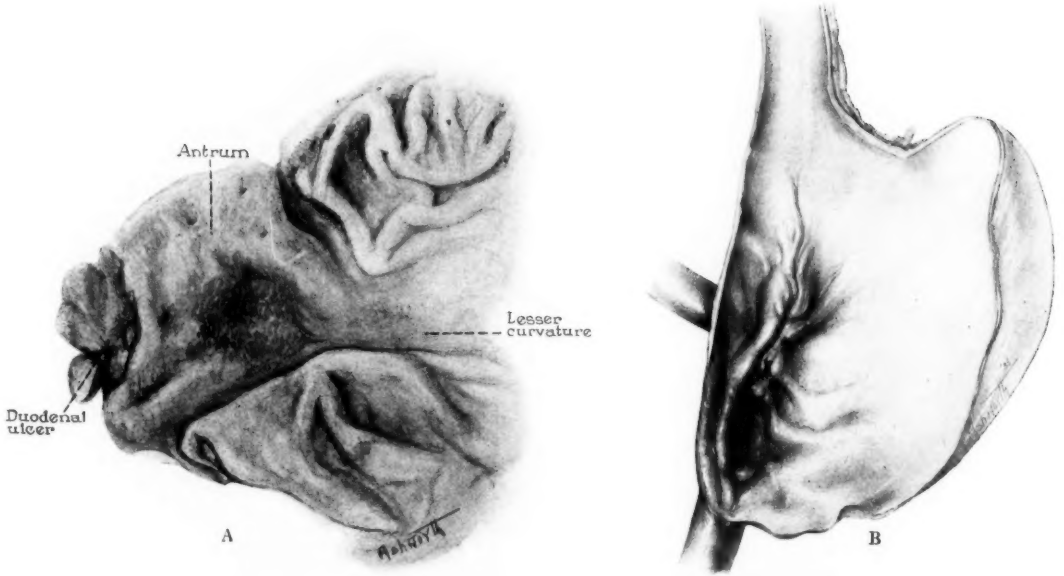


Fig. 20. This patient had been operated on by the author 7 years ago for an acutely bleeding duodenal ulcer. He was completely free from symptoms until he developed acute cholecystitis and died at another hospital after surgical interference. The patient indulged in hard liquor before and after the gastrectomy. A, Distal part of the stomach removed by operation showing duodenal and pyloric ulcer; also, severe gastritis. B, Postmortem specimen. Anterior wall of the remaining stomach has been removed. No gastritis found. This is a typical Billroth II stomach resection as popularized by Finsterer and presented in this paper.

The Surgical Treatment of Gastroduodenal Ulcerations.—M. E. Steinberg.

CLINICAL SURGERY

THE SURGICAL TREATMENT OF GASTRODUODENAL ULCERATIONS

The Technique of Gastric Resection

M. E. STEINBERG, M.D., Portland, Oregon

PARTIAL removal of the stomach is more important in the cure of ulcer than the removal of the ulcer itself. A partial palliative gastrectomy occasionally is indicated when a large callous ulcer is situated near the cardia; in spite of the fact that the ulcer is not removed, the patient obtains instantaneous relief. The cause of pain apparently is not always in the seat of the ulcer itself. In cases of long standing callous penetrating ulcers there may be certain periods of freedom from pain. Possibly, when a spasm of the motor part of the stomach takes place from a new attack of gastritis, increase in the acid value of the gastric juice, or other unknown causes, the symptoms may be aggravated.

The technical details and the various methods used in performing gastrectomy definitely affect mortality and morbidity. Functional results frequently are influenced by the anatomical relationships between the stomach and the jejunum. It is for this reason that every statistical study of results, especially regarding morbidity and mortality following stomach resection, should include a detailed description of the technique used.

It is not the purpose of this paper to present a historical background for the operation to be described. We should, however, not fail to mention Finsterer's contributions to the advancements made in surgery of peptic ulcer; in numerous publications he has described his modification, based on his wide experience, of the Billroth II operation.

Before a surgeon selects a method of procedure, he should familiarize himself with the evolution of stomach surgery and, through a thorough analysis of the literature, acquaint himself with the safeguards advocated as well as with the disappointments and errors of others.

Only few complications are unpredictable. What may appear as imponderable variations in technical details, unless carefully carried out, may assume major importance. An extra suture or two at the lesser curvature of the gastrojejunal anastomosis may not only protect this corner against an undue pull, but will cover a small terminal part of the stomach which may be devoid of its blood supply. I have observed 2 instances at postmortem examinations in which leakage from the proximal lesser curvature angle of the anastomosis occurred after gastrectomies performed by the chief surgeon of one of the well known European clinics. Too much suturing at this corner will be instrumental in producing an acute angulation with temporary stasis in the duodenum which may endanger the duodenal stump. On the other hand, too little suturing at the same corner may cause an angulation downward and allow the escape of stomach contents into an atonic and dilated duodenum. A hemorrhage from a smaller or larger blood vessel in the mesentery or in the line of anastomosis may cause troublesome or fatal complications. Thus we see that numerous details present themselves which are important factors in bringing about success or failure.

From the Emanuel Hospital.

TECHNIQUE

There should be no undue haste in the search for the offending lesion, neither should there be undue haste in the selection of the safest method of removal. Retreat after irreparable manipulations or proceeding to completion of operation with an unsafe or unsound method will prove equally fatal.

If no ulcer is found on inspection or palpation, a few blood vessels along the greater curvature, about 4 to 5 centimeters from the pylorus, are ligated and severed. Adhesions between the mesocolon and the posterior wall of the stomach are separated. The posterior wall of the stomach along the lesser curvature is inspected and palpated. The posterior wall of the duodenum is also

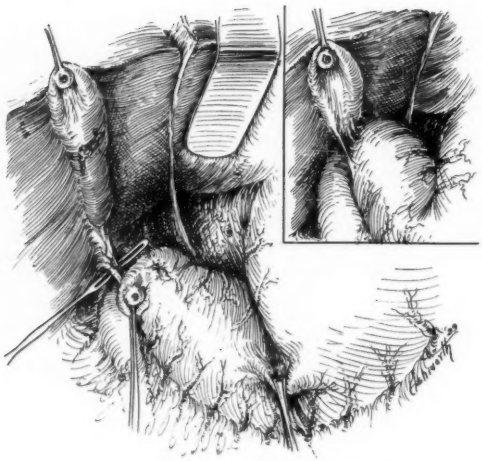


Fig. 1. Gall bladder released from adhesions to the anterior duodenal wall and the pylorus. Insert, gall bladder adherent to the ulcer on the anterior wall of the duodenum.

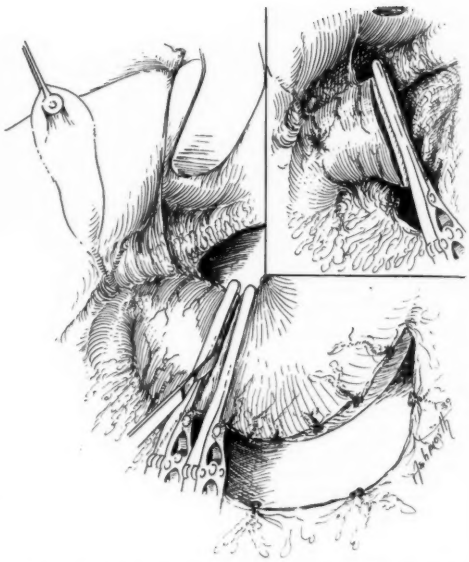


Fig. 2. Distal part of the stomach divided. Insert, the thick muscular layer incised down to the mucosa. Mucosa is eventually removed. This pyloric stump may be utilized for the author's pyloroplastic closure of the duodenum or for the Finsterer exclusion operation. With the clamp on the pyloric stump, the adhesions on the posterior wall of the duodenum may be divided with more convenience and the pyloric stump eventually removed if a typical closure of the duodenum is contemplated.

examined. The finding of a large ulcer penetrating into the pancreas may at once suggest the course of action to be taken. Large lymph nodes frequently indicate the presence of an ulcer, malignancy, or gastritis. The lymph nodes may point to the location of the ulcer. The avascular area in the gastrohepatic omentum may also be opened and the finger introduced along the lesser curvature and the posterior wall toward the esophagus in search of an ulcer. If no ulcers are found, an opening is made in the anterior wall of the stomach. The contents are aspirated and sponged and the hand is introduced for palpation. The anterior and posterior walls of the lesser curvature are examined. The use of wide retractors also helps in the inspection and palpation of the interior of the stomach. The index finger is introduced through the pylorus into the duodenum.

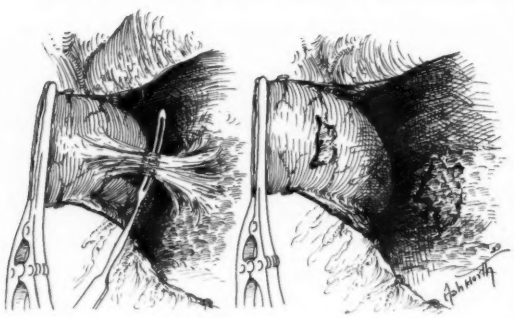


Fig. 3. Adhesions between the posterior wall of the stomach and the pancreas separated. This pyloric stump, after the mucosa is removed, may be utilized as the author's pyloroplastic closure of the duodenum or as a Finsterer exclusion operation with the mucosa removed. Or, it may be altogether removed and the duodenum closed in a typical manner.

One must be careful lest the finger be pushed through a fragile area of granulation tissue and penetrate through the posterior duodenal wall. Such an accident accounted for one of our fatalities. Even though this thorough examination reveals no lesion, it does not necessarily mean that none is present. On one occasion, the stomach was resected for a duodenal ulcer, and a good sized chronic ulcer was found on the lesser curvature although a thorough examination of the interior of the stomach had been negative. Recently a roentgenogram was negative for duodenal ulcer and none was found by the palpating finger introduced through the pylorus. An incision was made into the duodenum, however, and

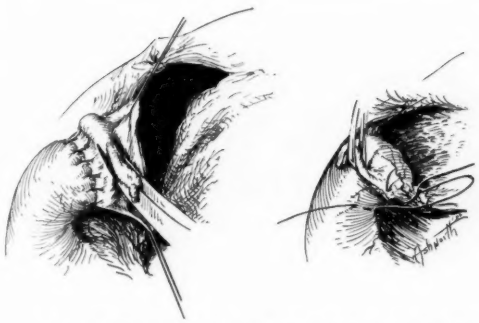


Fig. 4. The author's pyloroplastic closure of the duodenum. a, left, The serosa of the anterior wall of the duodenum which has been mobilized covers and tampons the ulcer on the posterior wall of the pancreas. The thickened part of the muscle of the distal pyloric stump has been previously removed. b, The distal pyloric stump, which has been flared open, and with the mucosa removed, is sutured over the anterior wall of the duodenum.

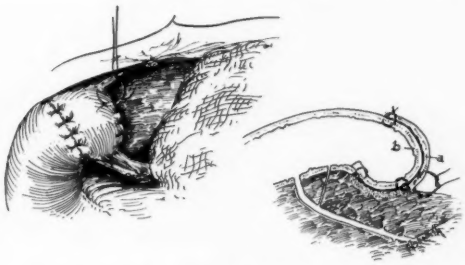


Fig. 5. The author's pyloroplastic closure of the duodenum. a, left, The pyloric stump flared open and denuded of its mucosa is sutured to the anterior wall of the duodenum. A few sutures are applied between the serosa of the wall of the stomach and the thickened pancreatic capsule. b, The cross-section view of the pyloroplastic closure of the duodenum plugging the ulcer bed penetrating into the pancreas.

a linear ulcer of the lesser curvature of the duodenum was revealed near the papilla, measuring about 2.5 centimeters. In still another patient the opening of the duodenum revealed an obstruction at the papilla. The x-ray examination revealed only an obstructive lesion at the outlet of the stomach.

When an ulcer of the duodenum is found, the important problem is to determine whether the ulcer can be removed safely. Such orientation depends on an adequate incision and the good exposure of the first part of the duodenum of the gall bladder, and, under certain circumstances, of the common duct. An ulcer adherent to the gall bladder or hepatic flexure more frequently may be dissected away from adhesions with safety. If an opening should be made in the anterior wall of the duodenum, it is possible to mobilize the duodenum and to use its anterior wall to secure a safe closure.

At this stage it may be found necessary to decide whether or not the gall bladder should be

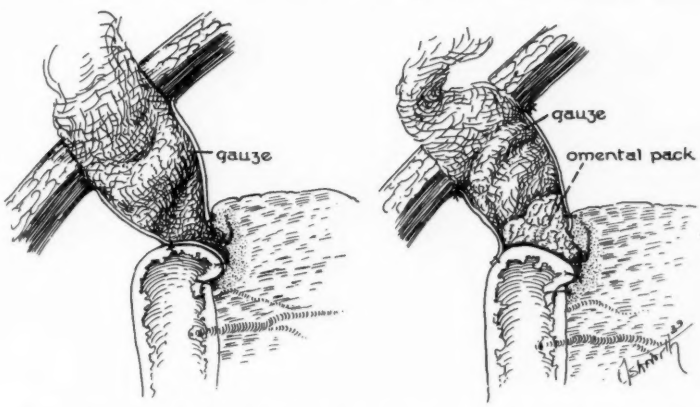


Fig. 6. The author's pyloroplastic closure of the duodenum in case of an acutely bleeding ulcer. a, left, The pyloric stump has been denuded of its mucosa and sutured to the peritoneum. The serosa of the anterior wall of the pyloric stump has been sutured to the anterior duodenal wall. Gauze packing applied to an acutely bleeding ulcer on the posterior wall of the pancreas. b, An acutely bleeding ulcer on the posterior wall of the duodenum packed, transfixed with an omental plug, and with gauze pack over the omental plug.

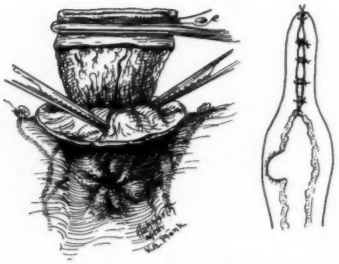


Fig. 7. a, left, Removing the mucosa of the pyloric stump in a Finsterer exclusion operation. b, Suturing of the denuded wall of the pyloric stump as practiced by Plenk.

Fig. 8. a, left, Opening made into the anterior duodenal wall for palpation, inspection, and removal of the duodenal contents. b, Inspection of the ulcer on the posterior wall of the duodenum. The ulcer on the posterior wall of the duodenum may be removed if the conditions are favorable. Otherwise, the author's pyloroplastic closure can still be utilized provided the duodenum is mobilized.

removed, since, buried in adhesions, it may not empty itself properly and thus later be responsible for residual symptoms. If the ulcer penetrates into the duodenohepatic ligament or into the posterior wall of the pancreas, or both, resection of the duodenum including the ulcer may injure the pancreas, accessory pancreatic duct, the common duct, and even the portal vein and the hepatic artery. Though the ligation of the gastroduodenal or the pancreaticoduodenal artery is practiced by some surgeons, occasionally it may result in a necrotic area of the pancreas, for the reason that these vessels give off branches to the pancreas.

The origin of the right gastro-epiploic artery also gives off occasional branches to the pancreas, and for this reason, the main stem or its branches should be ligated only along the greater curvature of the stomach.

It is apparent then, that attempts to remove ulcers which form thick and edematous inflam-

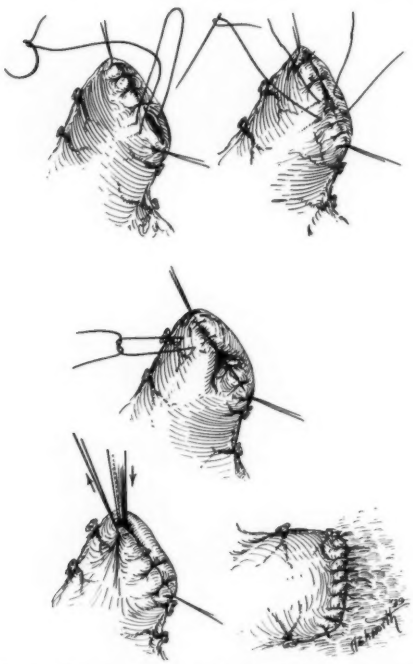


Fig. 9. Closure of the duodenum in a typical manner. The last row of sutures is taken between the anterior duodenal wall and the thickened pancreatic capsule.

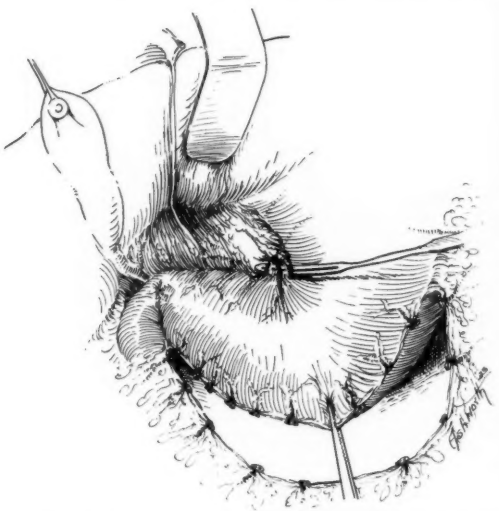


Fig. 10. The removal of the duodenal ulcer offers no difficulty. Blood vessels along the greater and lesser curvatures are severed down to the duodenum.

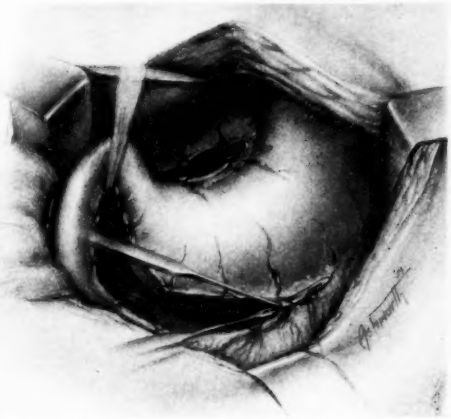


Fig. 11. The removal of the ulcers of the duodenum offers no difficulty. Blood vessels along the greater and lesser curvatures are severed and ligated. Diagrammatic.

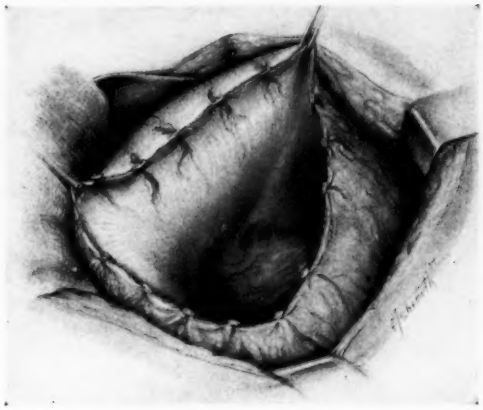


Fig. 12. The removal of the ulcers on the anterior or posterior wall of the duodenum offers no difficulty. Blood vessels along the greater curvature are ligated and severed.

matory masses will result in increased mortality because of leakage from the duodenal stump, because of pancreatitis, or because of injury to the other vital structures. Peritonitis, one of the frequent causes of death, is usually caused by leakage from the duodenal stump. It is difficult, therefore, to share the enthusiasm of some surgeons with much experience who make the claim that they are able to remove with impunity every ulcer of the duodenum. Since the anatomical and pathological realities are indisputable and the fatal result after such a procedure only too frequently reported in the literature, this optimistic claim is possibly based on fortuitous surgical material.

If it is found that the typical removal of the ulcer is not safe, one of three procedures may be used. If the ulcer is penetrating and there is still a possibility of mobilizing sufficient of the duodenum proximal to the ulcer, the duodenum may be closed and the ulcer left *in situ*. When the duodenal ulcers and the first part of the duodenum are buried in a mass of thick inflammatory adhesions, the Finsterer exclusion operation may be employed. There must be no dead spaces between the duodenal ulcer and any part of the excluded pylorus which will permit retention and possibly reflex stimulation of gastric secretion. Retention in the excluded stump also may cause leakage and fatal peritonitis. It is important to reiterate that exclusion of the ulcer alone does not always protect the patient against hemorrhage, perforation, or even persistence of symptoms. One of the patients succumbed from perforation of a duodenal ulcer following the exclusion operation and resection. Another had a hemorrhage and the symptoms persisted so that it was necessary later to remove the ulcer. Similar cases have been reported in the literature.

If the Finsterer exclusion operation is performed, at least the mucosa should be removed from the antrum so that the possibility of reflex stimulation of the secretion of gastric juice from the proximal remaining portion of the stomach may be reduced.

For the past 4 years in the majority of cases the author's *pyloroplastic closure* has been used in place of the Finsterer exclusion operation, with excellent results. Unlike the exclusion operation, pyloroplastic closure has no physiological objections. It permits direct attack on the ulcer and is especially valuable in the presence of acutely bleeding ulcer. Technically and anatomically it is feasible in the great majority of penetrating ulcers. The anterior wall of the duodenum, which is mobilized, covers and acts as a tampon to the bed of the ulcer where the bleeding vessel may be transfixed. A flap of the stomach wall denuded of its mucosa in turn is used to cover the anterior wall of the duodenum. A closure is thus made which is even safer than a typical one under the most favorable conditions. It is not necessary to expose the course of the common duct either in a typical Finsterer exclusion operation or in the pyloroplastic method. The common duct should be identified only in borderline cases when it has been decided to remove the ulcer in a typical manner.

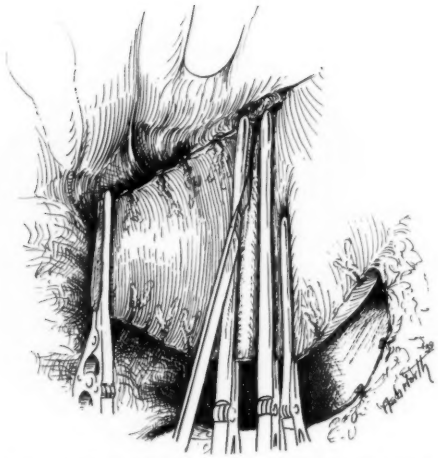


Fig. 13. The mid portion of the stomach is being removed. The distal part of the stomach has either been utilized for the author's pyloroplastic closure of the duodenum or has been removed as in the typical duodenal closure.

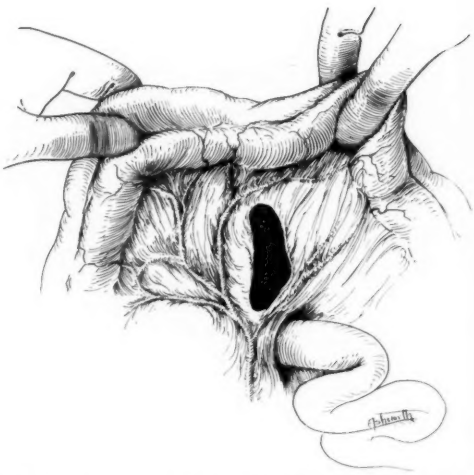


Fig. 14. An opening is made in the mesocolon close to the emerging loop of the jejunum.

The accompanying illustrations demonstrate some variations in the pyloroplastic method of duodenal closure.

In a doubtful case the following plan of action is adopted. A few blood vessels are ligated along the greater curvature of the stomach, about three finger breadths above the pylorus. The thin avascular area of the gastrohepatic mesentery is opened and the branches of the right gastric artery along the lesser curvature are ligated about the same distance from the pylorus as on the greater curvature. Two Payr clamps are applied in this area and the stomach is divided. The distal stump of the stomach can be utilized either for a Finsterer exclusion operation or the author's pyloroplastic closure. Even if the ulcer on the posterior wall or on the anterior wall of the duodenum can be removed in a typical manner, the distal stump of the stomach, if held taut, gives easy access to the adhesions between the duodenum and the pancreas. The division of the distal part of the stomach between two Payr clamps, as the first step, makes it possible to treat the duodenal ulcer by any method which appears to be safest for the patient. As soon as a decision regarding this most important step in the resection has been made, one proceeds with a typical resection.

The blood vessels along the greater curvature of the stomach are more numerous toward the pylorus. At about the middle of the greater curvature, near the fornix, the blood vessels are farther apart. Each blood vessel is separated and ligated three times. One ligature is applied close to the greater curvature and two ligatures close to the vascular arcade. The middle colic artery runs between the gastrocolic omentum and the transverse mesocolon. If the ligature carrier is introduced too deeply, there is danger of ligating the middle colic artery. Such an accident may take place when there are adhesions about an ulcer on the lesser curvature of the stomach which penetrates into the transverse mesocolon. One must then isolate the middle colic artery first before proceeding with the actual removal of the ulcer.

When the ligation of the blood vessels is made distal to the vascular arcade, the omentum may lose some of its blood supply and become dusky and cyanotic. Such an accident is to be feared particularly when the omentum is quite fat. The ligation of the blood vessels along the greater curvature is progressing toward the fornix. The extent of the resection along the greater curvature depends upon the condition for which the resection is performed. Resection for gastric ulcer or duodenal ulcer in older individuals, need not include much more than half of the stomach. When dilatation and hypertrophy are associated with a long standing obstruction, the hypertrophy takes place chiefly in the motor part of the stomach. The frequent mistake is to remove too little of the stomach. When operation is indicated for a duodenal ulcer in a young individual with high

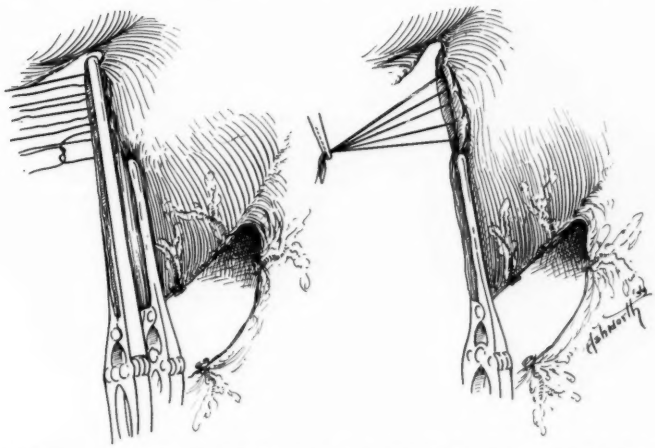


Fig. 15. a, left, The part of the stomach next to the lesser curvature is being closed by mattress sutures. b, The larger Payr clamp including the crushed part of the stomach has been removed. The anastomosis is made where the smaller Payr clamp is applied.

acidity or for jejunal ulcer, the resection should be more generous.

The gastrocolic ligament at the pyloric end of the stomach separates into two leaves. The anterior leaf leads to the greater curvatures of the stomach and the duodenum, and the posterior leaf leads to the posterior surface of the duodenum and the pancreas. The ligation of these mesenteries in one mass may result in troublesome bleeding and the danger of ligating not only the middle colic artery but also the pancreaticoduodenal artery and the origin of the gastro-epiploic artery. The posterior leaf should be ligated very carefully in order to mobilize the posterior wall of the duodenum and not to injure the pancreas. Usually numerous delicate arteries and veins are present which bleed freely.

The right gastric artery, as a rule, is of small caliber. It gives off branches to the distal part of the lesser curvature and the duodenum. This blood vessel is not always identified. Usually the distal part of the gastrohepatic omentum near the duodenum is ligated, by means of 2 ligatures proximally and 2 distally. The ligatures are transfixed since they are apt to slip and cause annoying hemorrhage. When extensive mobilization of the superior or greater curvature of the duodenum is desired, the small blood vessels along the superior border may be ligated at this time or at the beginning. These blood vessels originate either from the right gastric, hepatic, or the cystic vessels. The ligature carrier should not be introduced too deep, for if adhesions and anatomical variations are present, the important structures in the duodenohepatic ligament may be closer to the superior border of the duodenum. There is, therefore, the danger of ligating the common duct, the hepatic artery, or the portal vein. The ligation of the small blood vessels along the superior border of the duodenum, contrary to some opinions, does not greatly disturb the blood supply, particularly when one is sure to have sufficient of the anterior wall. The main blood supply to the duodenum comes from two parallel arcades along

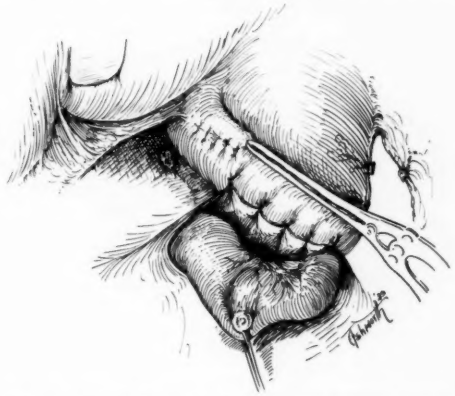


Fig. 16. The left leaf of the mesocolon is sutured to the posterior wall of the stomach.

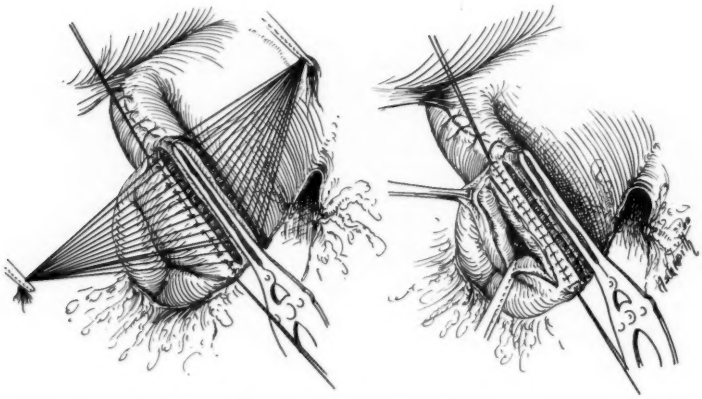


Fig. 17. a, left, The posterior wall of the serosal sutures in the jejunum and the posterior wall of the stomach have been applied. The wall of the stomach, anteriorally and posteriorally, has been incised down to the mucosa. The hemostatic sutures on the anterior and posterior walls of the stomach have been applied and are held by forceps. b, The jejunum has been opened and the hemostatic sutures cut.

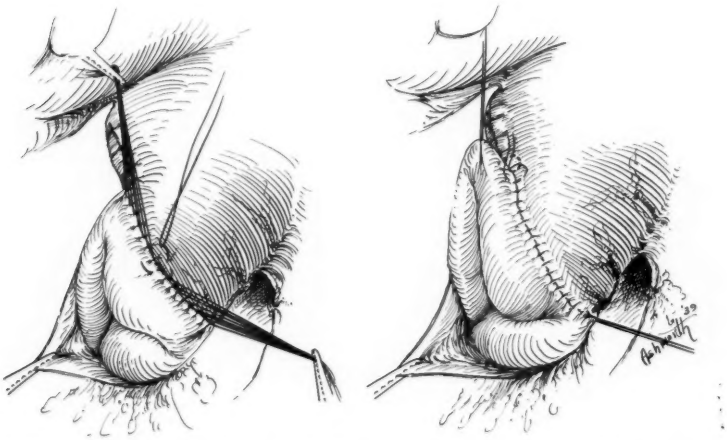


Fig. 18. Application of interrupted silk sutures to the anterior wall of the anastomosis.

the mesentery. To facilitate the ligation of the blood vessels along the superior border of the duodenum, one assistant retracts the liver and another pulls the pyloric stump downward and to the left. The strands of connecting tissue from the gastrohepatic omentum to the posterior wall of the duodenum and to the pancreas are separated at any step of the operation which appears convenient.

In a typical procedure a Payr clamp is applied to the pylorus, and then the ulcer, together with a narrow strip of the duodenum, is removed. It is not advisable to use crushing clamps on the duodenal stump. Under favorable conditions, when there is sufficient of the posterior wall of the duodenum left, a closure with a clamp is without difficulties. However, when the ulcer on the posterior wall of the duodenum or on the superior border penetrating into the duodenohepatic ligament has been removed, there is, as a rule, very little of the posterior wall left so that safe closure is difficult. A clamp applied crushes and de

Fig. 19. A diagrammatic view of the completed operation. With the omentum and the transverse colon raised out of the abdominal cavity, the anastomosis between the jejunum and the stomach assumes more often a position between the vertical and horizontal lines.

vitalizes the narrow available strip of healthy duodenum on the posterior wall and jeopardizes the procedure. The opening in the duodenum is closed with an interlocking hemostatic stitch of No. 00 chromic and two rows of interrupted silk. After the anterior wall of the duodenum has been freely mobilized, there is plenty of loose tissue to invert against the thickened pancreatic capsule. The realization that duodenal leakage is responsible for a certain number of fatalities should make one hesitate lest only an esthetic closure is performed. A "Moynihan" hypnotic stitch here and there or an occasional drain will save a life. Again it is important to emphasize that mobilization of the duodenum assures a safe closure of the duodenal stump even under the most difficult circumstances when there is very little of the posterior wall left. The posterior wall of the duodenum cannot be mobilized with safety in the presence of penetrating, inflammatory tumor masses.

The stomach, which has been separated from the duodenum, is reflected to the left. The first assistant retracts the left lobe of the liver and also pushes down on the pancreas. The left gastric artery and its branches are ligated and divided along the lesser curvature. When there is a great deal of fat, one must be careful not to penetrate the wall of the stomach. In such a case it is advisable by blunt section and palpation to introduce a hemostat so as to spread apart the fat along the lesser curvature. Frequently, particularly when a good part of the stomach is removed or when it is necessary to mobilize the lesser curvature more freely, the left gastric artery is ligated at the pancreaticogastric ligament. The ligation should be closer to the lesser curvature and should not be toward the celiac axis where there is danger of including a rare accessory hepatic artery which supplies the left lobe of the liver.

Four ligatures are introduced; 2 proximally, 2 distally. One of the proximal ligatures, which is made of chromic No. 1 catgut, is left long and grasped with a hemostat. There still remain several blood vessels on the anterior and posterior walls of the stomach which are running from the

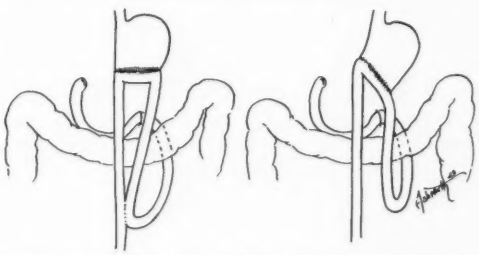


Fig. 21. a, left, An antecolic stomach resection which should give no trouble. b, An antecolic stomach resection which would favor stagnation of stomach contents in the proximal loop.

Fig. 22. A series of roentgenograms after Billroth II resections. These roentgenograms were taken about 3 to 4 weeks after the operation.

lesser curvature downward and outward. These are ligated and separated in order to free this part of the stomach for a safe closure or anastomosis. The omental tags hanging from the wall of the stomach are also removed. The ligation of the blood vessels mobilizes a great deal of the stomach wall and enables one to make the anastomosis without tension. It also robs the stomach of considerable blood supply and possibly helps to reduce the gastric acidity in the remaining part.

The stomach, which has been mobilized and freed from its blood supply and attachments in the manner described, is lifted upward and put on a stretch. Two large Payr clamps are applied to the stomach in the direction of the axis of the body so that the esophagus will be in the line of anastomosis which is made at the greater curvature. The clamp on the lesser curvature reaches about three finger breadths distal to the esophagus. A narrow strip of the stomach along the lesser curvature is closed by interrupted through-

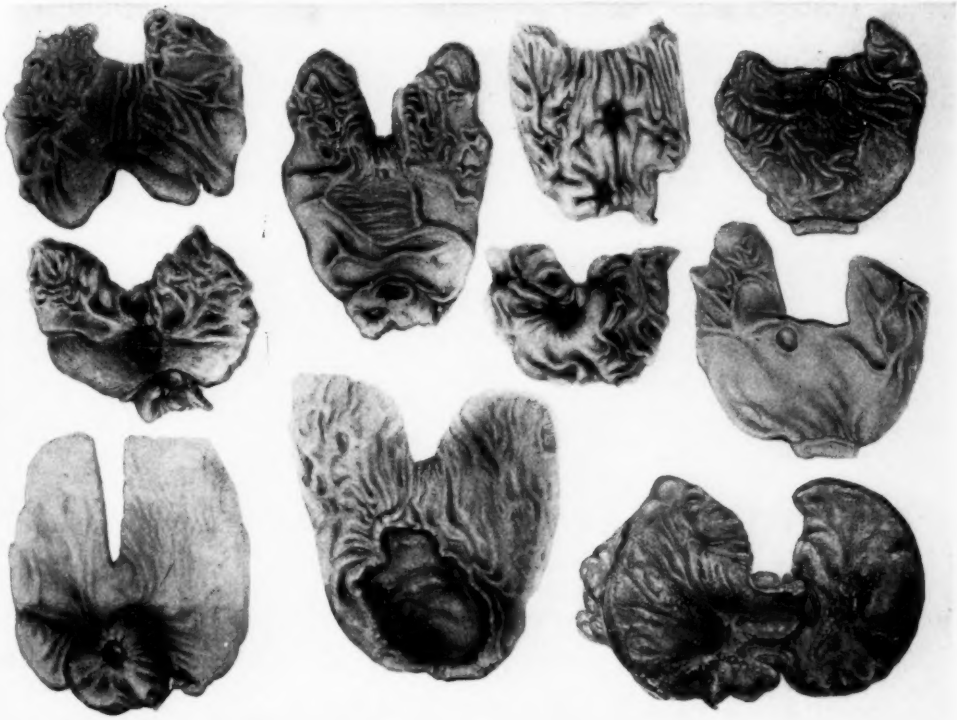


Fig. 23. A group of ulcers, some carcinomatous, removed in the reported series.

and-through linen sutures. A small Payr clamp is applied to the stomach along the greater curvature proximal to the large clamp. The large clamp is taken off, the crushed stomach wall is cut away, and the part of the stomach near the lesser curvature is inverted by interrupted silk sutures. A part of the gastrohepatic ligament including the ligated stump of the left gastric artery is sutured to the most proximal end of the lesser curvature of the stomach.

The transverse colon is lifted out of the abdominal cavity and the first part of the jejunum is identified. The right hand is introduced along the left side of the mesocolon and the left side of the spine, keeping in touch with the mesocolon. The loop of the small bowel which is fixed is the first part of the jejunum. This part of the jejunum, its emergence under the root of the mesentery, and its direction, must always be visualized. One must ascertain that there is not the slightest torsion of the jejunum before the anastomosis is made. Not infrequently there are adhesions between the jejunum and posterior wall of the mesocolon which distort the direction of jejunum and obscure location of the duodeno-jejunal angle. These adhesions must be separated.

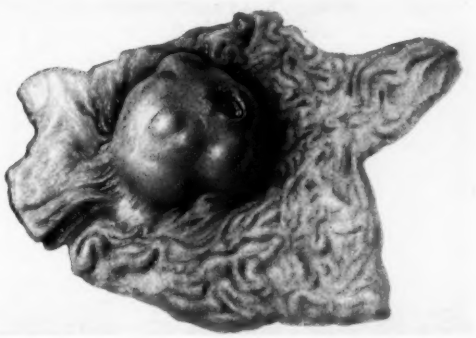
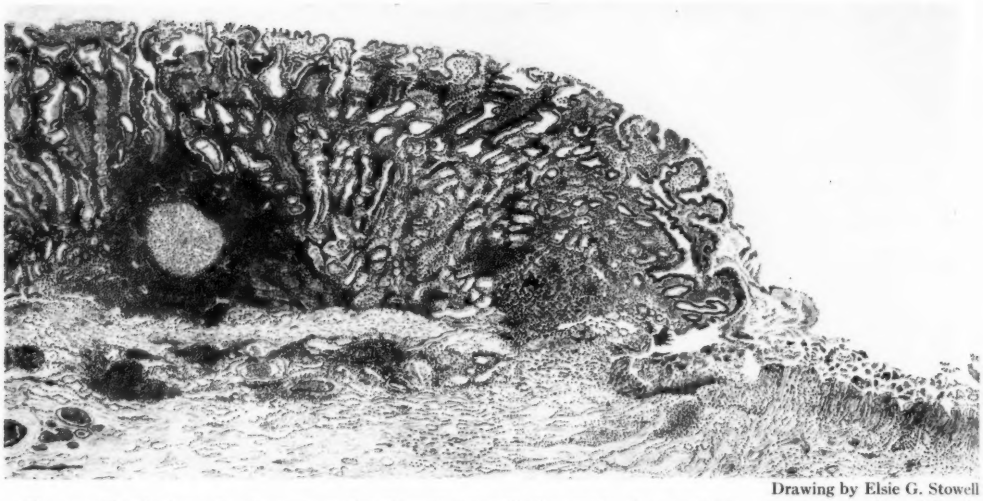


Fig. 24. Specimen removed by stomach resection; a deep gastric ulcer on the surface of a large myoma.



Drawing by Elsie G. Stowell

Fig. 25. Histologic study of a cross section through the wall of a gastric ulcer on the lesser curvature. Parietal cells at *A* are predominating and appear to be outside of the tubules. Such appearance is due to early digestion of groups of chief cells, and has been mistaken for carcinoma.

The opening in the mesocolon is made where the jejunum emerges from under the root of the mesentery. The incision is made in an avascular area. It should not encroach directly on the main vascular arcade. When the mesocolon is short from scarring, it is inadvisable to do a retrocolic anastomosis. Depending on the conditions one may perform either a Billroth I or an antecolic anastomosis. The transverse colon is replaced. The left leaf of the mesocolon is sutured to the posterior wall of the stomach about 2.5 centimeters from the line of anastomosis. On the lesser curvature the mesocolon is sutured for about 2 to 3 centimeters, and on the greater curvature for about 5 to 6 centimeters proximal to the anastomosis. The attachment of the mesocolon higher on the greater curvature keeps the anastomosis in a vertical axis. One must be careful not to injure the blood vessels of the mesocolon. When the stump of the stomach is small, only the left leaf of the mesocolon is sutured to the posterior wall of the stomach. The right leaf is then sutured to the jejunum.

The first serosal suture between the stomach and the jejunum is made of fine interrupted black silk. The jejunal loop should not be taken too close to the ligament of Treitz because the stomach pulls up higher under the diaphragm and causes dangerous tension on the line of anastomosis. On the contrary, the proximal loop should not be too long because it may sag and cause retrograde filling and stasis in the duodenum or proximal jejunum. The proximal jejunal loop should measure not less than 6 centimeters. In case a jejunal ulcer follows stomach resection, an edematous condition of the short proximal jejunal loop would make an end-to-end anastomosis very difficult. In 3 cases with jejunal ulcers the proximal loop was anastomosed to the distal one end-to-side and the gastrojejunal anastomosis was made distally.

The second row of sutures is made with chromic catgut No. 00 and is continuous. The serosa and muscularis along the posterior and anterior wall of the stomach, close to the small Payr clamp, are incised with a sharp knife down to the mucosa. Hemostatic sutures are applied to the individual blood vessels. The sutures are left long and held in two hemostats. The hemostatic sutures produce a puckering of the stomach wall. It is possible that it aids in establishing a mechanical control which prevents regurgitation of the bile and pancreatic juice into the stomach and prevents postoperative gastritis. The incision in the jejunum is made smaller than in the stomach. There are only a few blood vessels to be ligated. The small Payr clamp along the greater curvature of the stomach is taken off and the crushed part of the stomach wall is removed. The stomach contents are aspirated and a sponge is introduced into the lumen of the stomach. For the posterior suture line, which is taken through all the layers

chromic No. 0 catgut is used. Two or three additional interrupted sutures are introduced along the posterior wall to reinforce the continuous one. Also, the corners of the proximal and distal end of the anastomosis are reinforced with one or two additional sutures. The end sutures are always kept on the stretch to avoid kinking or torsion of the jejunum. Torsion of the jejunum or bulky suturing may be responsible for postoperative difficulties. On the other hand, hasty maneuvering may cause an occasional leakage and peritonitis. The same sequence of sutures is repeated on the anterior wall. Two or three silk sutures are used along the lesser curvature of the stomach and anterior and posterior walls of the jejunum to protect this corner against leakage and also to prevent regurgitation of the stomach contents into the duodenum. The anastomosis along the greater curvature is also reinforced with several fine silk sutures including only a narrow part of the stomach and jejunal wall since a kink is easily formed at the distal end of the anastomosis. One orients himself again as to the position of the proximal and distal parts of the jejunum and carefully inspects the posterior line of anastomosis. The right leaf of the mesocolon is then sutured to the anterior wall of the stomach about 2.5 to 3 centimeters from the line of anastomosis. It must be definitely established that the slit in the mesocolon at the lesser curvature is closed. The omentum and the transverse colon is lifted, and the jejunum proximal and distal to the anastomosis is inspected. Frequently a deep pocket occurs between the mesocolon on the right side and the proximal jejunum anastomosed to the stomach. Since the loops of the small intestine may herniate through this opening, it is advisable to close the opening provided no dangerous kinks are thus created in the proximal loop of the jejunum. The duodenal stump is again inspected and the abdomen is closed.

When the mesocolon is short and thick, particularly when it is shrunken from a penetrating ulcer, it is not advisable to use the typical retrocolic technique. In such cases the gastrectomy is completed by the antecolic method. There are some additional potential dangers of herniation in the antecolic type which may take place between the jejunal loops and the colon, or between the entero-anastomosis and the jejunal mesentery. These complications can be minimized by suturing the jejunal mesentery to the lateral anastomosis. The proximal loop of the jejunum is anastomosed at the greater curvature. Usually such a direction of the jejunum prevents rotation on its axis. On the other hand, if the proximal jejunum is anastomosed to the lesser curvature, there is more chance of its being partially rotated on its axis. If in addition to some torsion of the jejunum the anastomosis is made bulky and not straight and directly opposite the mesentery, there may result troublesome stasis. When antecolic anastomosis is made with the proximal jejunal loop anastomosed to the greater curvature, the stomach should be severed on its axis and not in the manner used in a typical retrocolic operation in which it is cut in the axis of the body. This is of some importance because if a part of the stomach is closed along the lesser curvature, the stomach contents will invariably pass into the proximal jejunal loop and into the duodenum and this may result in troublesome symptoms particularly if no entero-anastomosis is provided for. Dyspeptic symptoms resulting will disappear only after several months when the wall of the duodenum and proximal jejunum become hypertrophied and compensate for the anatomical error.

The proximal jejunal loop anastomosed to the stomach should not be short because when the stomach pulls up under the left diaphragm, it might cause tension with danger of leakage and a disturbance of blood supply from the pull on the mesentery. A short proximal jejunal loop may be kinked at its emergence under the transverse colon and omentum when the short stump of the resected stomach is pulled up under the diaphragm. The jejunal loops between the stomach and the entero-anastomosis should be of equal length; otherwise, stasis may result from a redundant and kinked loop. For good drainage, the entero-anastomosis should be placed between the most dependent part of the proximal jejunal loop and the corresponding distal loop. The entero-anastomosis should not be too small and should be made in a straight line opposite the corresponding mesenteries.

ANESTHESIA

Previously infiltration and anterior splanchnic anesthesia where used in a considerable number of resections. At the present time local anesthesia alone is used in a rare case. The night before the operation the patient receives 1.5 grains of nembutal. In the morning, 2 hours before operation, 6 grains of sodium amytal are given by mouth. One-half hour before the operation 1/6 grain of morphine and 1/150 grain of atropine are given. Not infrequently the operation is started under local anesthesia. Ether is given when necessary. When the patient is apprehensive, ether is administed at once with nitrous oxide gas induction. The abdominal wall is

blocked off with 0.5 per cent novocain in every case. After the abdomen is opened, the peritoneum on each side of the midline is infiltrated with novocain. The transverse colon is delivered to the outside and the transverse mesocolon along the root of the mesentery ballooned out with the novocain solution. The mesentery along the duodenum and the right and left gastric arteries and the right and left gastro-epiploic arteries are infiltrated and ballooned out. After the stomach is skeletonized, the capsule of the pancreas is also infiltrated with the anesthetic solution. The patient is not under deep anesthesia at any time during the operation. Though it takes from 3 to 5 or even 6 hours to perform the operation, if it happens to be a complicated jejunal ulcer, thus far we have seen no shock in any of our cases. The barbiturates allow a liberal amount of novocain which in turn blocks the pathway to the higher centers. The extra time spent is compensated by a smooth convalescence. Caffein and sodium benzoate are always administered at the completion of the operation.

PRE-OPERATIVE AND POSTOPERATIVE TREATMENT

Only those patients who have complications receive elaborate care. If there is no obstruction, the stomach is never washed before the operation. If there is an obstructive lesion, the blood urea is determined, and, if there is indication of kidney damage, the patient is treated accordingly. The stomach is washed out several times a day, glucose and saline are given, and also blood transfusions. In a rare case when the patient is extremely emaciated from a prolonged severe obstruction, and if the condition does not improve, a jejunostomy should be performed before the stomach resection is done. Jejunal feeding and other supportive measures must be instituted in order to improve the general condition of the patient and also the kidney function. Blood transfusions are given to anemic or emaciated patients.

After the patient has sufficiently awakened from the anesthetic, the head of the bed is elevated. As a routine measure, the patient receives 2000 cubic centimeters of 5 per cent glucose in water and 1000 cubic centimeters of normal saline each day for the first 2 days. A Levine tube is introduced into the stomach and the contents are aspirated every hour. This tube may be removed during the first or second day for 2 or 3 hours or more at a time. As a rule, no tube is used on the third postoperative day. As soon as the patient is removed from the operating room, he is turned several times and, after he awakes, the nurse is instructed to support the incision carefully so that the patient can cough up tenacious mucus and take deep breaths. These respiratory exercises are repeated every half hour or every hour. As soon as possible, the patient is encouraged to exercise his legs and arms to improve his general circulation. Prostigmin is given 6 hours before the operation, every 4 hours during the first 24 hours, and every 6 hours during the second 24 hours. No troublesome postoperative distention of any kind has taken place following a gastrectomy.

Water and sweetened tea, jello, and zwieback are given beginning with the third day. The food is gradually increased so that on the fifth and sixth days the patient receives cooked cereals, puddings, and such.

COMPLICATIONS

Peritonitis. Peritonitis rarely takes place from outside contamination unless the abdominal wound becomes disrupted. Since no clamps are used in the performance of a gastro-intestinal anastomosis, more or less spilling of the gastro-intestinal contents takes place in every gastrectomy. Piercing the needle through the whole thickness of the stomach or intestinal wall also involves a certain amount of bacterial contamination. Thus far, no peritonitis has resulted in over 200 gastro-intestinal anastomoses from such soiling. This is clinical evidence of the resistance of the peritoneum against a certain degree and type of bacterial invasion.

The commonest causes of peritonitis are the following: (1) leakage from the duodenal stump —(a) the removal of callous penetrating ulcers of the duodenum forming inflammatory tumor masses, (b) from faulty closure, particularly through the failure to mobilize the anterior duodenal wall, (c) the use of crushing clamps on the duodenal stump, (d) escape of pancreatic juice from the ulcer base or injured pancreas with the digestion of the devitalized wall of the duodenum, (e) escape of bile from the injured common duct, (f) ligation of the gastroduodenal, pancreaticoduodenal, or right gastro-epiploic arteries at its origin, (g) ligation of a functioning accessory pancreatic duct; (2) leakage from faulty anastomosis—(a) necrosis of the gastro-intestinal wall through poor blood supply, strangulation of tissues, or suturing of crushed tissues, (b) poor hemostasis, (c) anastomosis under tension which may produce a pull on the line of suturing or stretch the blood vessels in the mesentery, (d) kinks, torsion, and faulty mechanics ("vicious circle"), (e) inadequate pre-operative and postoperative treatment; (3) the use of Finsterer's

exclusion operation in the presence of duodenal obstruction. Drainage should be instituted when the closure of the duodenal or pyloric stump appears insecure.

Subphrenic abscess. Subphrenic abscess can be prevented in the same manner as peritonitis. Drainage should be used in case of poorly controlled hemorrhage or insecure duodenal closure.

Vicious circle. It is outside of the scope of this paper to discuss the many possibilities which masquerade under the rather vague term of "vicious circle." The proper interpretation of all the technical factors concerned and the physiological principles involved will make a so called "vicious circle" after stomach resection an extremely rare occurrence. The use of the Levine tube and the proper pre-operative and postoperative care usually allows the emptying of the stomach contents so that small feedings can be started on the third postoperative day.

Hemorrhage. The use of two knots on every blood vessel left in the mesenteries, also, individual ligation of every blood vessel along the line of the anastomosis, should prevent hemorrhage.

Pulmonary. No fatal respiratory complications occurred in this series of cases in spite of the fact that there were a number of gastrectomies performed on patients in advanced age suffering from anemia and obstruction.

The following measures are used to prevent respiratory complications: maintenance of proper nutrition and vitamin supply; also, water and mineral balance before and after the operation; blood transfusions in anemic or debilitated patients; frequent breathing exercises and deep coughing to bring up tenacious mucus; immediate and frequent change of position; vigorous exercises of arms and legs; avoidance of deep prolonged anesthesia; the use of an operative technique which is meticulous in every detail in order to avoid suturing and strangulation of large masses of tissue, hematoma, and abscess formation.

Retention of gastro-intestinal contents unrelieved for a considerable period of time with resulting inanition may also predispose the patient to pulmonary complications.

Late sequelæ. No operative procedure in which the stomach is anastomosed to the jejunum can absolutely exclude the possibility of jejunal ulcers unless the acid gastric juice can be completely eliminated. From experimental laboratory and clinical experience, several conditions have been discovered under which jejunal ulcers may take place. Some authors believe that there are certain types of jejunal ulcers which are surgically incurable (Mandl). Finsterer, however, states that radical and adequate surgery can cure every jejunal ulcer. In spite of the most radical surgery there will occasionally be a patient whose acid gastric juice will not be sufficiently controlled to prevent a jejunal ulcer. Fortunately, in my own experience such cases are rare.

In order to prevent jejunal ulcers, the resection must be a subtotal one, particularly when the acid values of the stomach contents are high and when the individual with a duodenal ulcer is young. When the stomach is dilated in the presence of a chronic obstruction, the usual mistake is to remove too little of the stomach. Kinks, torsion, or a small gastro-intestinal anastomosis is responsible for the retention of the acid gastric contents and predisposes to jejunal ulceration. An entero-anastomosis does not altogether prevent the alkaline duodenal juices from passing the gastro-intestinal anastomosis. A "Y" anastomosis, however, sidetracks the alkaline pancreatic juice and bile and is a contributing factor in producing jejunal ulcers.

If a "Y" anastomosis becomes necessary when an operation for a jejunal ulcer is done in the presence of a short, edematous proximal loop, I have placed it proximal to the gastro-intestinal anastomosis.

Finsterer's exclusion operation with the mucosa left intact has been responsible for two jejunal ulcers in this series of cases.

The modified von Eiselsberg exclusion operation, which is known in this country as the Devine operation, in the greatest majority of cases will result in jejunal ulcers. This has been demonstrated experimentally by the author and by surgical end results.

Pressure symptoms. Few patients complain of pressure symptoms immediately after a heavy meal. These symptoms may persist for a few months or more but have rarely been serious or disabling. Relief is obtained after the patient lies down for a few minutes. Some reflex phenomenon, which takes place in the stomach and which is explained on the same basis as palpitation complained of by people with an intact stomach, may be responsible for these complaints. It may also be an exaggeration of the splanchnic reflex which takes place in numerous individuals after a heavy meal and which is characterized by dizziness and a desire to sleep or lie down. This splanchnic reflex will take place more readily and violently in an individual with a resected stomach since the food empties into the jejunum rapidly.

Gastritis. Many of the continental surgeons have blamed gastritis and jejunitis for most of the dyspeptic symptoms which take place after stom-

ach resection. It is my opinion that some of the symptoms are due to faulty technique, inadequate surgery, disabling adhesions, or improper indications for operation. The presence of bile and pancreatic juice which may stagnate in the stomach may be a causative factor of gastritis and of some of the dyspeptic symptoms. Stagnation in the proximal jejunal loop or duodenum, in the presence of a previous malfunction, should also be considered as a probable cause of dyspepsia. If the puckering sutures of the stomach are used, there is the possibility that a little or no bile enters the stomach.

It must be frankly stated that the cause of some of the residual symptoms following gastrectomy have thus far not been explained to our complete satisfaction. Fortunately, these complaints are not common and rarely serious.

REPORTED CASES

Since 1924 stomach resection has been adopted as a routine procedure for the relief of gastroduodenal ulcers. Thus far, 117 primary operations have been performed. Three patients succumbed to the operation. The analysis of the case reports discloses that each of those patients was admitted to the hospital on account of severe hemorrhage.

Contact has been made on several occasions with over 90 per cent of the surviving patients. By far the great majority of these patients are free from symptoms. They do not follow any specific diet and not a few indulge in liquor and smoking in spite of admonitions to the contrary. Five patients upon whom we performed gastrectomy complain of dyspeptic symptoms but consider themselves free from the original trouble.

Brief summaries of the cases of patients who have not been benefited by operation follow:

An Italian, male, aged 40 years, operated on in 1925 for duodenal ulcer, has bitterly complained on many occasions of obscure pressure symptoms after eating. He has not been seen or heard from for several years.

V. L., male, aged 48 years, Case A-6849, was operated upon, on April 11, 1936, for duodenal ulcer with complete obstruction. A Finsterer exclusion operation, without the removal of the mucosa from the excluded part of the stomach, was performed. This patient gained in weight and got along nicely until a year ago when he presented himself with a history of hemorrhage and typical symptoms of jejunal ulcer. He was operated upon again at the Emanuel Hospital on December 4, 1938. The excluded part of the stomach was removed, the jejunostomy opening was closed, and the remaining stomach reduced in size. A Finney-Haberer anastomosis was substituted for the Billroth II modification performed previously. This patient has had no complaint since his last operation and has gained 30 pounds.

R. W., male, aged 53 years, Case A-8096, operated upon February 15, 1939, for duodenal ulcer. An exclusion operation was performed with the removal of the mucosa but the ulcer was left *in situ*. The Finney-Haberer gastroduodenal anastomosis was used in this case. This patient improved but had some vague gastro-intestinal complaints and some pain. Finally he developed a severe hemorrhage. The symptoms were due to the ulcer which had not healed and to duodenal stasis which was not discovered at the time of the original operation. At the second operation, performed May 27, 1939, part of the pyloric stomach and the duodenal ulcer which penetrated into the pancreas were treated by the author's pyloroplastic closure. Since the acid values of the stomach were high, only a very small part of the stomach was left and the operation was completed with a typical Billroth II modification (Finsterer). The patient thus far has made a good recovery. He still complains, however, of some dizziness and palpitation after eating.

G. K., female, Case E-6598, operated upon July 8, 1937, for a large penetrating ulcer of the duodenum. A Finsterer exclusion operation was performed. The patient was free from symptoms for a short period of time and then began to complain of pain relieved only by food. X-ray examination and symptoms pointed to jejunal ulcer. Since this patient had a large non-toxic goiter and a parathyroid adenoma, she was operated upon June 30, 1938. A subtotal bilateral thyroidectomy was performed including the removal of a parathyroid adenoma the size of a hazelnut. The patient showed freedom from symptoms for several months and then gradually began to complain of pain which could be relieved only by morphine. She was operated upon November 6, 1939. A large jejunal ulcer penetrating on the posterior wall of the peritoneum was removed. The excluded part of the stomach next to the duodenum was also removed. The duodenum was mobilized under the root of the mesentery on account of duodenal ileus. The duodenal stump was closed. The proximal end of the jejunum was anastomosed to the distal end and to the side, and the open end of the proximal loop was brought out as a temporary jejunostomy. The remaining proximal stomach was anastomosed to the wide anterior wall of the duodenum. The patient thus far was made a splendid recovery, and up to date is completely free from her symptoms.

Summaries of 3 cases in which patients succumbed.

Mr. X., aged between 40 and 50 years, was operated upon in a small private hospital for a large bleeding ulcer on the posterior wall of the duodenum which had perforated into the pancreas. The ulcer was treated by the author's pyloroplastic closure with a Finsterer's modification of the Billroth II. The patient expired 5 days after the operation. The postmortem examination revealed that the technical procedure was in good order. No blood was available for transfusion in that hospital and the after-care was not under my personal supervision.

Mr. H. R., Case number 83491, aged 69 years, was operated upon because of a bleeding gastric ulcer on the lesser curvature. At the operation it was found that there was also an ulcer in the duodenum. The patient had four transfusions before the operation on account of anemia. Finsterer's modification of the Billroth II was performed. The resection was used in this case on account of suspicion of malignancy. The patient made an uneventful recovery and on the day he was ready to go home, he expired suddenly. The postmortem examination revealed degeneration of the heart muscles with marked coronary sclerosis and thrombosis and occlusion of the coronary artery.

L. J., Case number A-8156, male. This patient had violent hemorrhage and received seven transfusions during

his stay in the hospital. He was operated upon March 23, 1939. A large ulcer on the posterior wall of the duodenum was found penetrating into the pancreas with a narrow passage to the duodenum. A typical Finsterer exclusion operation with removal of the mucosa was performed. This patient lived only 2 days after operation. The postmortem examination revealed pancreatic necrosis and considerable bile-stained fluid in the abdomen. In the attempt to palpate the ulcer the finger was thrust through the ulcer and the fragile posterior duodenal wall. This was not discovered during the operation on account of the posterior location of the ulcer and adhesions covering the ulcerated area. If the author's pyloroplastic closure had been used, the accident would have been discovered during the operation.

SUMMARY

The technique of the Billroth II stomach resection is presented.

One hundred and seventeen primary stomach resections were performed with a mortality of 2.6 per cent. Every patient who succumbed to the operation came into the hospital on account of severe hemorrhage.

Technical errors and faulty pre-operative and postoperative treatment are accountable for most of the fatalities.

The greatest number of gastrectomized patients are in excellent health. Those few patients who have not gained in weight and also those few who complain of mild dyspeptic symptoms all consider themselves cured of their original ulcers.

Thus far, there have been 3 definite failures. Two patients were operated upon for jejunal ulcers following the Finsterer exclusion operation with the mucosa left intact. A third patient who also had had the Finsterer exclusion operation performed developed a hemorrhage from the old duodenal ulcer. All 3 patients recovered from the secondary operations and are well at the present time.

The operation of gastric resection should become more popular provided that cases are carefully selected and that the knowledge and training of the surgeon are commensurate with the magnitude of such a procedure.

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TECHNIQUE OF PARAVERTEBRAL ALCOHOL INJECTION

Methods and Safeguards in Its Use in the Treatment of Angina Pectoris

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IN 1925 the Austrian surgeon, Felix Mandl, proposed paravertebral infiltration of procaine around the upper thoracic sympathetic ganglia for the relief of intractable angina pectoris. He claimed that procaine alone often sufficed to quiet the attacks over many days or even weeks. Unfortunately this long duration of relief has not been generally corroborated.[1] A year after Mandl's article Swetlow demonstrated that procaine block could be safely supplemented with ethyl alcohol. The effectiveness of this method of treating the severest forms of angina pectoris has now been attested by a large number of favorable reports (6, 13, 16, 19, 23, 26). Since our first experience in 1927, 85 paravertebral injections with alcohol have now been performed on 63 patients with various forms of medically intractable cardio-aortic pain, at the Massachusetts General Hospital. In 5 of these cases injection was performed by Dr. W. J. Mixter, in the remainder by myself. As a result of observations made on this large series and the follow-up reports of patients after upper thoracic sympathectomy (24), we are now convinced that pain arising from all varieties of coronary and valvular disease, as well as from syphilis of the aortic arch, will respond to either surgical or chemical destruction of the upper thoracic ganglia or the rami which join them with the heart and posterior spinal roots (Fig. 1). The reason why the results of these procedures are so far superior to the various forms of cervical sympathectomy is that all cervical operations fail to interrupt the direct thoracic cardiac nerves. These important accessory connections were first described a decade ago by Braeucker in Germany, by Jonnesco and Enarchesco in Roumania, and in this country by Kuntz and Morehouse. The experiments of White, Garrey, and Atkins (25) showed that cardiac pain experimentally produced in dogs was not relieved by resection of the inferior cervical and first thoracic ganglia, but was no longer felt when the lower pathways were included.

Unfortunately, most of the sufferers from severe coronary pain cannot safely be submitted to either a thoracic sympathectomy or a posterior rhizotomy. In order to help the majority of patients with advanced angina pectoris, it is therefore necessary to destroy these structures chemically by paravertebral infiltration. In our experience failure to relieve precordial and arm pain[2] by this method has never been encountered in the presence of signs of an effective paralysis of the upper thoracic sympathetic fibers, i.e., vasodilatation and sudomotor paralysis of the upper extremity and a Horner's sign. While miosis and ptosis are desirable, as they indicate a thorough infiltration of alcohol well up along the lateral border of the first thoracic vertebra, the production of a Horner's sign[3] is not essential for an effective block of the cardiac afferent fibers. The *sine qua non* is a clear-cut paralysis of nerves to blood vessels and sweat glands of face, neck, and upper extremity.

Inasmuch as accurate paravertebral injection of alcohol is difficult and a number of serious complications have been reported to me from other clinics, it seems advisable to describe the technique of injection which has been evolved at the Massachusetts General Hospital during the past 12 years.

TYPE OF PATIENT TO SELECT FOR INJECTION

Patients have been recommended for paravertebral injection by the cardiac consultants after a thorough trial of medical treatment has failed to bring adequate relief of severe anginal pain. The only requisite has been really severe and frequent bouts of pain. No patient has been refused injection because of recent coronary infarction or threatened cardiac failure.

The etiological factor for the anginal attacks has included arteriosclerotic and hypertensive heart disease, syphilitic aortitis, rheumatic heart disease with aortic regurgitation (1), and mitral stenosis. The oldest patient submitted to alcohol injection was 85 years old, the youngest 17. Twenty-six persons presented clear evidence of previous

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[1] After many attempts with procaine alone I have never seen it fail to stop cardiac pain for from 24 to 48 hours, but after this period anginal attacks have recurred with their usual frequency.

[2] One patient with reference of severe pain to the face, head, and neck has required secondary resection of the superior cervical ganglion.

[3] In this series available records show a clear-cut Horner's sign in less than half of the patients injected. Yet all patients who retained a hot, dry hand had lasting relief of cardiac pain. If the hand became moist and cool within the course of a few days, the attacks invariably recurred.

coronary occlusion, and 18 suffered from angina decubitus.

It has been our opinion that alcohol injection should be reserved exclusively for the poor risk patient with intolerable pain. With injection there is a 10 to 20 per cent chance of failure from lack of accurate infiltration, and about a 10 per cent chance of producing a troublesome chemical neuritis of the intercostal nerves. In the severe case neuritis is a minor matter in comparison with the relief of anginal pain, but the victim of milder attacks is less likely to tolerate the disagreeable neuritic sensations with equanimity. In general it may be stated that the severer the suffering and the poorer the operative risk, the more suitable is the patient for alcohol injection. If the less severe cases of angina pectoris are to be treated by cardiac denervation, it should be by actual operation.[1]

When patients with the most severe forms of angina pectoris are selected for operation, there is an appreciable danger in even such a minor procedure as paravertebral alcohol injection. Patients and their families should be warned of this before injection is undertaken. It has been our practice to state frankly that there are about 8 chances in 10 that the patient will have satisfactory relief from his cardiac pain. He should also be told that he is likely to have some transitory intercostal neuritis, and that there is 1 chance in 10 that it will bother him for several months. The risk of alcohol injection is far less than that of any other surgical procedure, but the technical difficulty of accurate injection prevents its being 100 per cent effective. If the injection fails to give sufficient relief, it can be repeated. As a final resort, the greater risk of surgical denervation may have to be faced in rare instances.

Finally, the physique of the patient makes a considerable difference in the ease of accurate injection. There should be very few failures in the thin-chested subjects with prominent spinous processes and transverse processes only a few centimeters beneath the skin. Here the needles need be inserted no more than 5 or 6 centimeters, but in the formerly heavily muscled and subsequently obese type exemplified by one ex-circus strong man, the needles may have to be thrust in to their entire length of 10 centimeters. It is well to select the former type for one's early attempts, but thereafter patients must be taken as they come, and the short-necked, stocky variety will unfortunately lower the record of successful results.

[1] Cervicothoracic operation is procedure of choice in this clinic for the patient who presents no unusual surgical risk, provided he has a relatively normal electrocardiogram and is free from syphilitic or active rheumatic heart disease. I believe that resection of the inferior cervical, first, second, and third thoracic ganglia through an anterior supraclavicular incision is the most satisfactory procedure yet devised. This approach, in which the anterior scalene muscle is divided, was devised by Gask for sympathetic denervation of the arm in Raynaud's disease. It gives a distinctly better exposure and can be carried out more rapidly and with less risk to the patient than the posterior approach through the head of the first or second rib.

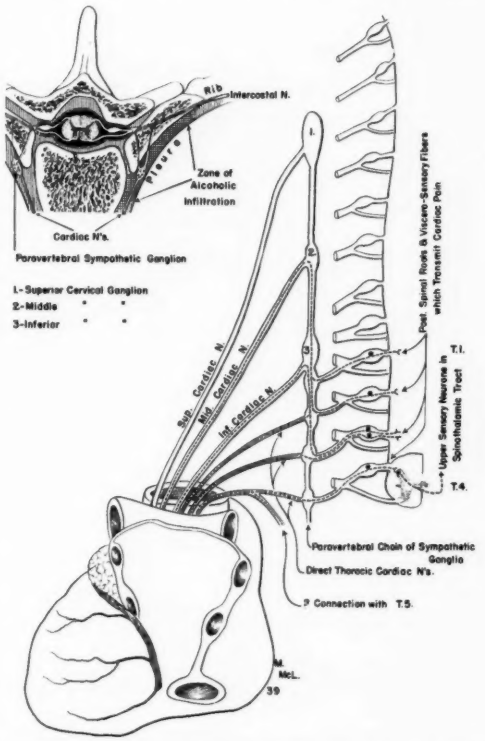

Fig. 1. The sensory nerves of the heart. Afferent fibers from the vagus are not shown, because they play no rôle in cardiac pain. There are possibly afferent fibers in the superior cardiac nerve, but if present they must establish connections with the cranial and upper cervical nerves rather than descending in the cervical sympathetic trunk (see Davis and Pollock). Although a thoracic cardiac ramus is shown arising from the fourth thoracic ganglion, it is questionable how often it exists.

ARTICLES REQUIRED FOR INJECTION

Needles should be 10 centimeters long and constructed of rustless flexible steel. Thin lumbar puncture needles or the special Labat needles sold by the Anglo-French Drug Company of New York are most satisfactory. Each needle should be equipped with a depth marker to measure the distance it is to be pushed beneath the rib. A short length of narrow rubber tubing or a bit of bone wax serves this purpose well.

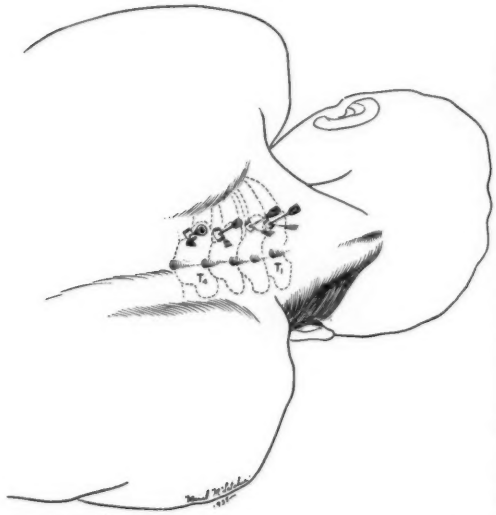

Fig. 2. Position of patient for paravertebral injection of the cardiac nerves. (From *Diseases of the Coronary Arteries and Cardiac Pain*, edited by Robert L. Levy, Macmillan, New York, 1936.)

A hypodermic needle for raising cutaneous wheals at the points of insertion of the larger needles.

Any good 5 to 10 cubic centimeter glass syringe which fits the needles will serve for injection.

A metal centimeter rule.

Solution of 1 and 2 per cent procaine hydrochloride without epinephrine.

Ninety-five per cent or absolute ethyl alcohol (C.P.).

A 1 cubic centimeter ampoule of lipiodol.

TECHNIQUE OF INJECTION

Paravertebral injection is performed on the side of the severest pain and against the anterolateral borders of the upper thoracic vertebræ. In 76 per cent of our cases the pain has been largely on the left side, in 11 per cent predominantly on the right side, and in 13 per cent it was bilateral. When bilateral, injection should be done on both sides with a few days' interval between. Whether more than the upper four thoracic ganglia and their rami ever require injection is not yet fully established, but it has been our practice to inject also beneath the fifth rib, and occasionally beneath the sixth if the pain is referred to an unusually low level. If the attacks are very atypical, it is advisable to do a preliminary injection with procaine alone and then exercise the patient to the point which would previously have induced an attack. In the typical case, however, this preliminary test is unnecessary.

Alcohol injection for angina pectoris should be carried out with the patient in his own bed, and preferably in an operating room, although with the aid of a nurse it can be undertaken in the patient's home. Injection in bed is not difficult for the operator, who is seated on a low stool, and it enables the patient to be kept quietly on his side until the alcohol has been fixed in the tissues. The injection must be performed without a general anesthetic, because this masks the evidence of a successful placement of the needles. The patient must therefore be carefully medicated, in order to enable him to lie for an hour on his side with the minimal amount of discomfort and emotional strain. Having seen 2 patients die of coronary thrombosis a few hours before the time set for injection, and 2 others develop infarction during the procedure, we have become sensitized to the danger of psychic strain in persons with severe forms of angina pectoris. Patients are therefore routinely given 3 grains (0.2 gram) of phenobarbital the evening before, and this is repeated once or twice in the morning before the bed is moved to the operating room. This usually ensures a drowsy patient. In addition it has been shown by Weiss that the barbiturates reduce the chance of toxic reactions to procaine. It is also safer to order 1/100 grain (0.6 milligram) of atropine sulphate subcutaneously as a protection against syncope and other vagal reflexes. If on arrival in the operating room the patient is still restless and worried, an additional 1/6 to 1/4 grain (10 to 15 milligrams) of morphine should be given before the needles are inserted. In any event morphine should be available in a syringe for immediate subcutaneous injection in case the patient develops severe pain during the course of the procedure.

The patient is laid on his side, upper back and shoulders close to the edge of the bed, legs drawn up, head flexed forward and supported on a thin pillow so that there is no lateral curvature of the cervical spine (Fig. 2). It is also important to make sure that the patient is not bearing too much weight on his lower arm, and to have both hands exposed so that they can be observed for changes in circulation and sweating.

The technique of inserting the needles is essentially Labat's second method of paravertebral injection. The bony landmarks for insertion of the needles are the spinous processes. As these are imbricated downward like shingles on a roof, the first rib and transverse process lie lateral to the seventh cervical spine (the first prominent process). This relation holds over the upper thoracic vertebræ. Procaine is injected intradermally 4

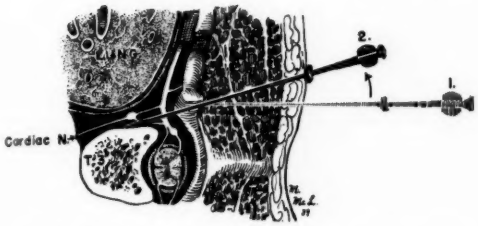

Fig. 3. Insertion of needle for injection of cardiac nerves. *1*, Needle inserted 4 centimeters to left of spinous process and tip in contact with transverse process of vertebra. Depth marker has been set at a point 3 centimeters from the skin. *2*, Shank of needle has been rotated outward and tip worked inward until at an additional depth of 3 centimeters it lies in contact with the side of the vertebra and in close approximation with the ganglionated sympathetic chain.

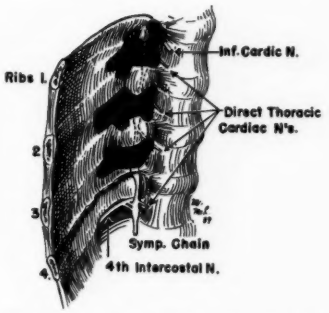

Fig. 4. Distribution of 2 cubic centimeters of methylene blue injected in the manner described in the text against the sides of the upper three thoracic vertebræ in a cadaver. The dissection shows the way solutions diffuse in the retropleural plane around the sympathetic ganglia, communicant rami, cardiac, and intercostal nerves.

centimeters lateral to the seventh cervical and upper three thoracic spinous processes.[1] The 10 centimeter needles (with depth markers on the shafts) are then inserted at these points and pushed inward perpendicularly to the skin until the transverse process or the articulating portion of the rib is touched at an average depth of from 2 to 5 centimeters (Fig. 3, first position of needle). It is important to visualize the depth of the ribs in order not to penetrate the pleura and puncture the surface of the lung. If this happens, a spontaneous pneumothorax occasionally develops in the course of a few hours. Once contact has been made with bone, the tip of the needle is manipulated caudad until it touches the lower border of the transverse process. The depth marker is then pulled out to a distance of 3 centimeters from the skin. Each needle is now inclined to an angle of approximately 20 degrees with the median sagittal plane and perpendicular to the curvature of the back in relation to the long axis of the thorax. When thrust inward on this bearing a second contact is usually made with bone at a further depth of 3 centimeters (Fig. 3, second position of needle). If sooner, the needle must be withdrawn and re-inserted at a slightly lesser angle. On the other hand, if no contact is made at 3 centimeters, the needle must be directed further toward the midline. The paravertebral ganglionated chains lie at an average depth of 3 centimeters beneath the transverse processes, running along the anterolateral surface of the vertebral bodies and looping over the heads of the ribs. The farther forward the tips of the needles can be inserted and still maintain their contact with bone, the less alcohol will come in contact with the intercostal nerves and the greater amount will surround the gray visceral rami which run forward from the sympathetic trunk to the heart. A useful trick in working the tip of the needle forward alongside the vertebra is to start with the bevelled tip pointed medially. When bone is touched, the tip of the needle can often be made to scrape along it if the needle is rotated through 180 degrees so that its bevelled tip is turned away from the bone. A depth of even 4 centimeters beneath the transverse process is quite safe, provided the tip of the needle still rests against bone. An infiltration in this region will diffuse freely through the retropleural space, bathing the spinal nerves, the sympathetic trunk and its rami, and the cardiac nerves which run anteriorly in the posterior mediastinum (Fig. 4).

In performing these injections, the needles should never be attached to the syringe. Care should be taken that the tip of a needle does not lie within the pleural space, in a blood vessel, or in an outward prolongation of the subarachnoid space. None of these eventualities is dangerous, provided it is recognized and the position of the needle corrected. With the tip touching bone, it is almost impossible for it to lie within the pleura. Rapid inspiration of procaine placed in the butt of the needle, or a cough reflex on injection, indicate that the tip lies within the pleural cavity. If it lies within a blood vessel or the subarachnoid space, aspiration of blood or spinal fluid will make these complications obvious. Bloody taps are frequent under the upper two ribs because the intercostal branch of the costocervical artery parallels the first and second thoracic ganglia. Spinal fluid is more rarely aspirated, but the possibility of a

[1] Following sterilization of the skin with tincture of iodine, acriflavine applied with a fine cotton applicator forms a jet-black color and is an excellent medium for marking these points.

Fig. 5. This roentgenogram illustrates a satisfactory position of an injection mass. After infiltration with procaine and alcohol, 0.3 cubic centimeter of lipiodol was injected through the uppermost and lowest of the four needles. There is good diffusion of the solution at the sides and in front of the bodies of the upper five thoracic vertebræ. A year and a half after injection this patient maintained complete freedom from his former severe attacks of right-sided angina pectoris.

high spinal injection of either procaine or alcohol is a serious matter. This is most likely to happen if the needle is passed over the upper border of a rib in a cephalad direction. I have withdrawn spinal fluid twice, and know of three instances of intrathecal injection of either procaine or alcohol. When all of the needles have been properly placed, they should form a characteristic pattern with their shafts lying in the same sagittal plane. The uppermost should be inserted deepest and point in a slightly more caudal direction than the others.

As soon as the needles are in position, 2 cubic centimeters of 2 per cent procaine should be injected into each. If properly placed, this minimal amount of solution produces clear-cut signs of intercostal and sympathetic nerve paralysis within a period of 5 to 10 minutes. Anesthesia appears in the axilla and over the third and fourth ribs front and back. No anesthesia develops over the first and second ribs, as this area is also innervated by descending branches of the third and fourth cervical nerves. No anesthesia should develop in the arm or hand, but this entire region, as well as the side of the neck and face, should become hot and dry. This unilateral sympathetic paralysis is particularly striking when the hands are cold or sweaty from nervousness. Horner's syndrome is a less useful sign, as it is often hard to make out with the patient lying on his side and with the pupils constricted after morphine. When these signs appear rapidly it is good evidence that the needle tips lie close to the sympathetic trunk. If they fail to develop within 10 minutes it is best to withdraw the needles and reinsert them at a later time. It must be borne in mind that procaine diffuses through the tissues far more readily than does alcohol, and experience has shown that unless a clear-cut block can be produced by a minimal quantity there is no assurance of a lasting paralysis with 5 cubic centimeters of alcohol.[1]

When satisfied that the needles have been placed correctly and that there is no anesthesia of the ulnar nerve or evidence of subarachnoid block, it is best to inject a further 3 cubic centimeters of 1 per cent procaine into each. This supplementary infiltration is to ensure a widespread anesthesia, so that the final injection of alcohol will be painless. The additional dilution of the alcohol does not seem to prevent an effective destruction of nerve tissue.

The final injection of 95 per cent alcohol is carried out very slowly; a total of 5 cubic centimeters is injected through each needle, but the plunger is drawn back after each half centimeter has run in to make sure that the needle tip cannot have shifted and penetrated a blood vessel or the subarachnoid space. All together several minutes should be spent in injecting the alcohol through each needle. If the patient complains of any undue discomfort, the injection must be stopped for a few minutes until the pain subsides. By following the alcohol with a few drops of lipiodol, the exact location of each needle can be identified by subsequent anteroposterior and lateral spinal x-ray films (Fig. 5). This is of great value in developing the technical skill of the operator and in finding the cause of failures. Often it is found that the highest point of injection is alongside the second thoracic vertebra, but the nerves at the side of the first are by far the most important. In the thick-backed individual it is often difficult to

[1] Five cubic centimeters of alcohol injected into the thigh muscle of a rabbit causes an area of necrosis little over a centimeter in diameter.

identify the first rib. As it is most undesirable to inject alcohol above it in the region of the brachial plexus, one is often forced to play safe and select the lower of two doubtful points. On several occasions when anginal attacks have persisted in the arm and under the upper sternum, subsequent x-ray films have shown no lipiodol above the second thoracic vertebra. This residual pain has disappeared after a more accurate injection under the first rib, made possible by x-ray localization.

After the needles have been withdrawn it is best to keep the patient on his side with his back supported by a pillow and as quiet as possible for an hour or more in order to minimize diffusion of the alcohol. He may then be shifted over onto his back, and after 2 hours allowed to assume any desired position in bed. Most patients can be up on the following day and leave the hospital within 72 hours.

COMPLICATIONS

The following early complications have been observed in this clinic.

Pleuritic pain has been troublesome in 4 patients within a few hours of the injection. This has appeared as the procaine has been absorbed, and it is surprising that it is not of more frequent occurrence. One, or at most two injections of morphine and chest strapping have invariably given satisfactory relief.

Severe pleuritic pain developed during injection in one case. This was probably caused by alcohol leaking into the pleural cavity. It necessitated large doses of morphine, but subsided within 6 hours.

Pneumonia followed injection in an 85 year old woman who was dying of coronary infarction. Injection in her case was undertaken because of the unusual intensity of pain, which had not yielded to large doses of opiates.

Pneumothorax has appeared within a few hours after injection in 2 patients. The cause of this is penetration of the pleura and puncture of the lung, so that air continues to leak from the injured alveoli for a number of hours. In one asthmatic patient aspiration was necessary for the relief of dyspnea.

Although no instance of intrathecal injection has occurred in this series, I have withdrawn spinal fluid on one occasion, and have always worried over the possibility of this most serious complication. The precautions which can be taken to avoid it are three: Never slide the needle over the upper border of a rib in a cephalad direction; always insert the needle detached from the syringe; and draw back on the plunger at frequent intervals during the actual injection. In spite of these precautions, a colleague has informed me that he has produced a high spinal anesthesia in the course of his preliminary procaine injection. However, if a volume of only 2 cubic centimeters of 2 per cent solution is used as a test for the position of each needle, the risk of injecting this small amount (40 milligrams) is practically negligible. Provided no abnormal anesthesia develops and the needles are not shifted, the danger of infiltrating alcohol intrathecally must indeed be very slight. Molitch and Wilson have recorded a Brown-Séquard paralysis, which came on almost immediately after a paravertebral injection of alcohol beneath the first rib. Fortunately the patient subsequently recovered from her paralysis and was relieved of her anginal pain. One other such accident has been reported to me. This also occurred following injection for angina pectoris, but no details are available.

Another serious early complication is coronary infarction. This has occurred twice in my experience during the course of injection with fatal results, and was probably the cause of an unexplained death which took place on the following day (see section on deaths to follow). As has been stated, similar accidents have resulted in 2 other patients a few hours before the time set for injection. John Hunter, an illustrious sufferer from anginal attacks, said that his life was in the hands of anyone who made him lose his temper. His sudden death, which took place after an argument at a medical meeting (9) bore out the truth of this remark. If serious coronary insufficiency in the form of either angina pectoris or coronary thrombosis can be precipitated by anger, it is equally likely to be brought on by the emotional strain of a surgical procedure. There is no way to predict this catastrophe, but much can be done to reduce its likelihood by careful preliminary medication, by doing injection with patient in his bed, and by taking care to avoid pain and emotional stimuli.

Late complications have been caused by intercostal irritation and neuritis. The sympathetic ganglia lie so close to the intercostal nerves that alcohol infiltrated around the chain cannot help bathing their trunks. They are paralyzed at first, but anesthesia begins to disappear in their anterior divisions within a fortnight. Within a month the intercostal nerves are recovering along their entire length, and with this there is a greater or lesser degree of hyperesthesia of the chest wall, which commonly persists for a number of months. Most patients state that pressure of clothing irritates the tender skin and that there is a burning sensation with occasional shooting pains. In most cases the discomfort is quite bearable and clears

up in a month or two. In others, about 10 per cent, it is more troublesome and requires mild sedation with acetyl salicylic acid or empirin compound, phenobarbital at night, and occasional doses of codeine. Baking the hypersensitive areas is often a great help. With the exception of a neurotic woman and one other individual in whom the injection failed to relieve the anginal attacks, the patients have all stated that they would willingly submit to a second injection if their attacks should ever recur.

There is no question but that neuritis constitutes a serious objection to treatment by alcohol injection. In advanced coronary disease its disadvantages are far less than the risk of mortality from operation, but it prevents the application of the method to any but the severer cases of angina pectoris.

DEATHS

Five patients have died within 2 weeks of injection:

CASE 1. An 85 year old woman, already moribund from coronary infarction, but with an extraordinary amount of precordial pain, was injected and her cardiac pain relieved. Three days later she died of pneumonia.

CASE 2. A 62 year old lawyer with generalized arteriosclerosis suffered from a rapidly increasing number of attacks of typical angina pectoris—up to 20 in a single night. Electrocardiogram showed abnormal T-waves in the first and fourth leads, consistent with coronary disease affecting chiefly the anterior surface of the left ventricle. After 3 weeks' hospitalization without benefit from medical therapy, paravertebral injection was performed. Just as the infiltration of alcohol was completed he suffered a syncopal attack with fall in blood pressure to 40 millimeters systolic, and a reduction in pulse rate from 120 to 60. His respiration nearly ceased. After oxygen inhalation and stimulating drugs the blood pressure slowly rose to 100/70 millimeters. On the 2 succeeding days he developed a fever of 101 degrees and a leucocytosis of 24,600. During the next 12 days he ran a slowly downhill course, with progressive heart failure and electrocardiographic changes typical of extensive coronary thrombosis. Postmortem examination revealed both old and recent infarction of the myocardium.

CASE 3. A 65 year old man gave a history of angina pectoris for 10 years, on an arteriosclerotic basis. Two and a half years previously he had had an attack of coronary thrombosis followed by bouts of cardiac pain of increasing frequency and severity. His pain was limited to the right precordium and arm. The electrocardiogram indicated the existence of coronary disease. Paravertebral injection was undertaken after he had failed to respond to medical treatment. The infiltration of alcohol was accomplished without any pain or other complication, but 10 minutes afterward his speech became confused and his color cyanotic. He rapidly lost consciousness. No pulse or heart sounds could be detected. Ten minutes later he was dead. Permission for an autopsy could not be obtained.

CASE 4. A woman of 56 with syphilitic aortitis had suffered from shortness of breath and attacks of substernal pain for 3 years. In spite of thorough antiluetic treatment her anginal attacks had increased in severity and frequency over the past 18 months. For 3 weeks she had been in bed with pain in the precordium and both arms, which was precipitated by nervousness, eating, or the least exertion. Nitroglycerine produced an extremely unpleasant sense of fulness in her head. Her blood pressure was 160/50. There were definite enlargement of the heart, broadening of the aortic arch without aneurysm, and signs of advanced aortic regurgitation without congestive failure. Paravertebral injection was done on the patient's left side in her home in a distant city. The injection was carried out uneventfully, and 3 hours later I left her in apparently unchanged condition. Her local doctor wrote that during the night she awoke, sweating profusely and vomiting. There was one attack of pain in her right chest, but none on the injected side. Cyanosis developed, with increasing shortness of breath and a fading radial pulse. She died 20 hours after injection. No autopsy was done, but it seems most probable that her death was caused by a painless coronary occlusion.

CASE 5. A fifth fatality occurred in a younger man of 36 with rheumatic heart disease. He suffered from angina pectoris decubitus secondary to destruction of the aortic valve. In addition he had an enlarged heart, orthopnea, signs of mitral as well as aortic valvular disease, and evidence of active rheumatic infection. Paravertebral injection was undertaken because he used up to 100 nitroglycerine tablets each 24 hours and still failed to obtain adequate rest. It was felt that although he was a distinctly poor risk his recovery was unlikely unless he could obtain relief from the constantly recurring attacks of pain in his left arm and precordium. Injection of alcohol was uneventful, but he complained afterward of an unusual amount of chest pain, which required strapping and morphine. The pleuritic pain lasted 2 days, and although not very severe seemed to exhaust his remaining cardiac reserve and to precipitate decompensation. His discomfort and the signs of decompensation seemed to improve, but he died on the tenth day with sudden cessation of the heart beat. Postmortem examination revealed a moderate amount of free fluid in both pleural and pericardial cavities, but no evidence of any thrombosis or myocardial infarct. The usual local reaction to the injection of alcohol (edema and beginning fibrosis) was noted in the retropleural tissue around the upper thoracic sympathetic ganglia and their rami.

WHAT HAPPENS TO THE WARNING SIGNAL IN THE PATIENT WITH CORONARY DISEASE WHEN PAIN IS REMOVED?

Sir James Mackenzie made the statement, so frequently quoted, that if the surgeon could remove the pain of angina pectoris it would be harmful, because the patient would have no danger signal to warn him when he was overtaxing his heart. This prediction has fortunately not been borne out in our personal experience.[1] We have seen a number of patients after bilateral paravertebral injections who have had no residual pain, but they have always known when they were having an attack. In the absence of anginal pain the warning signal has consisted of certain well defined symptoms such as dyspnea, palpitation, a peculiar clutching sensation in the suprasternal

[1] While an adequate warning sign is preserved after alcohol injection, this is not always the case after procaine, which diffuses much more widely through the posterior mediastinum and seems to produce a complete cardiac denervation, vagal as well as sympathetic. Before we realized this, one man developed an alarming attack of paroxysmal dyspnea, having been allowed to exert himself beyond his ordinary tolerance.

notch, or flushing of the skin. When the pain pathways have been interrupted on one side only, patients have often noted much milder, but typical attacks on the other side. To my knowledge there has been only a single case in which no warning signal has persisted after injection. This has been reported to me by the surgeon who performed the injection, and resulted in the death of the patient, who refused to limit his activity.

Far from being harmful, surgical relief of intolerable and frequent attacks of angina pectoris is often a life-saving procedure, a point emphasized in Miller's recent book on *Angina Pectoris*. This is especially true when recurrent nocturnal attacks prevent sleep. Two patients in this category were so worn out by lack of sleep that their attending physicians believed they were going to die of exhaustion. Both were relieved of pain by paravertebral injection. One lived for 5 years, until fatal decompensation followed his advancing syphilitic aortitis. The other, injected in a distant city, returned to his work as a storekeeper and remained well until an ill advised attempt was made to drain his chronically inflamed gall bladder 9 months later. This induced sudden death on the operating table. Numerous case histories could be cited in which the patients have been bed ridden for months on account of pain secondary to coronary thrombosis or aortic regurgitation with rheumatic fever. Relief of pain has enabled them to get out of bed and leave the hospital. One of the rheumatic group subsequently became self-supporting as a seamstress.

Although an adequate warning signal is nearly always preserved, patients must be warned that their cardiac reserve will not necessarily be increased. They must continue, therefore, to limit their activity to a level within the tolerance of their coronary circulation. Whatever they are doing must be stopped when telltale shortness of breath or any peculiar sensation develops, and they should continue to use nitrites as before injection.

DURATION OF RELIEF

That freedom from severe anginal pain may last indefinitely after paravertebral injection with alcohol is surprising, in view of the fact that after injection of the branches of the trigeminal nerve neuralgia is seldom relieved for more than 6 months. In this series complete interruption of pain has now lasted for over 2 years in 14 cases, and for more than 5 years in 3. One patient has remained over 90 per cent relieved of his former unbearable pain for 8 years. The reason for this long lasting paralysis of the cardiac pain fibers is explained by their anatomical structure. The alcohol acts on the delicate rami which unite the sympathetic ganglia with the intercostal nerves dorsally and run ventrally as even more delicate strands to the heart. Whereas the white and gray rami are rarely larger than a millimeter or two in diameter, the cardiac branches are often no thicker than a hair. In addition, the sensory axones which they contain are protected by little or no myelin. They are therefore more easily penetrated by alcohol than the peripheral nerves, which are thicker and are covered by heavy sheaths of fibrous tissue. An alternative possibility is the fact that even the severest cases of angina pectoris may have spontaneous remissions. When this occurs a successful block, even if of short duration, may suffice to tide the patient over his period of intractable pain.

Return of troublesome paroxysmal pain after successful injection has been observed in only 5 of the patients in this series. In 2 the recurrent attacks have been so mild that they have been easily controlled by routine medical treatment. In only 3 have they recurred with their previous intensity after intervals of 3 years to 5 months. One of these has been submitted to a second injection. This patient, an unusual case in whom cardiac pain occurred during attacks of paroxysmal auricular fibrillation, has had a second recurrence of rapid irregular beats associated with the anginal pain. Repeated injections of alcohol seem to be less effective, due no doubt to the conversion of the loose retropleural areolar tissue into scar.

ANALYSIS OF STUDY

In this paper on the technical aspects of paravertebral alcohol injection for the relief of cardiac pain, there is no need to report the results achieved by the method in detail. This has been done in a number of previous articles (1, 23, 24, 26). To bring the series up to date the results are summarized in Table I. When allowance is made for the type of patient dealt with, it is evident that paravertebral alcohol injection, in comparison with surgical denervation, carries the minimal risk and with only a slight sacrifice in effective results. However, injection must be performed with the most scrupulous technique in order to place the alcohol with sufficient accuracy to ensure destruction of the sympathetic rami and the cardiac nerves. As mentioned, animal experiments have shown that 5 cubic centimeters of alcohol produces an area of necrosis not much over 1 centimeter in diameter. Observations made during two postmortem examinations have shown that this applies to man. The infiltration of alcohol must therefore be far more exact than when

TABLE I.—RESULTS IN 62* PATIENTS WITH INTRACTABLE ANGINA PECTORIS TREATED BY PARAVERTEBRAL ALCOHOL INJECTION.

	Per Cent
Complete or nearly complete relief of pain	52
Reduction of severe attacks to a mild form which can be treated satisfactorily by routine medication	30½
Unsatisfactory result	9½
Died within 2 weeks of injection	8

*One patient left for South Africa within 10 days of injection and has been lost track of. The others have been followed over periods ranging from 2 weeks up to 8 years.

procaine is used, as this drug diffuses so much more widely through the retropleural tissues. It is always evident when the alcohol has been correctly placed, because of the unequivocal signs of paralysis of the upper thoracic sympathetic rami. When these signs persist, pain is just as effectively relieved as though the same structures had been resected.

Surgical resection of the inferior cervical and upper thoracic ganglia, or posterior rhizotomy of the corresponding spinal nerves,[1] are unquestionably better methods of dealing with cardiac pain than is paravertebral alcohol injection, provided the patient can tolerate an operation of such magnitude. Surgical denervation causes no neuritis and destruction of cardiac afferent pathways is nearly certain, but many of the severest sufferers from angina pectoris are impossible risks for surgery, and even in the most carefully selected group there will be an occasional fatality.

In choosing between surgical and chemical denervation much depends on the training of the surgeon. Cutting the posterior spinal roots or cervicothoracic ganglionectomy falls within the routine operations performed by the neurosurgeon. But the technique of paravertebral alcohol injection is more difficult to learn and requires frequent repetition to perfect. It can be acquired by anyone experienced in the use of regional anesthesia by practice in the postmortem room with methylene blue (Fig. 4). Experience in human cases is most readily acquired by the use of paravertebral procaine block as a test for the degree of vasoconstrictor tone. This form of diagnostic injection, described by White (22), may be used routinely in testing patients with Raynaud's disease and thromboangiitis obliterans to determine whether the peripheral circulation can be improved by sympathectomy. In this way surgeons who are connected with a large clinic for the study of peripheral vascular disease can carry out paravertebral injection at frequent intervals. Satisfactory results can be obtained in no other way, nor can really serious complications be avoided. Blocking the cardiac nerves with alcohol should therefore not be attempted by anyone who has occasion to perform paravertebral injection only once or twice a year.

[1]Section of the upper five thoracic posterior spinal roots has been performed by Loyal Davis, Grant, and Cone, with uniform success. It should be a particularly valuable procedure when paravertebral alcohol injection has failed and has produced a troublesome neuritis.

SUMMARY AND CONCLUSIONS

1. This paper describes the technique of paravertebral injection of the upper thoracic sympathetic rami and the cardiac nerves in patients with intractable angina pectoris.

2. The value of chemical versus surgical destruction of the cardiac sensory nerves is compared.

3. It is concluded that in experienced hands paravertebral injection carries the minimal risk and is therefore applicable to the greatest number of patients who suffer most severely from cardiac pain.

4. Technical difficulties should prevent an effective destruction of the cardiac fibers in not more than 20 per cent of cases. Whenever there has been a persistent vasomotor and sudomotor paralysis, anginal pain has disappeared. The freedom from attacks is usually permanent, and the patients maintain an adequate warning signal.

5. The type of patient most suitable for injection, the dangers and complications which have been encountered are discussed. The chief of these is alcoholic neuritis of the intercostal nerves.

6. It must be reiterated that alcohol block of the cardiac nerves cannot be expected to give satisfactory results except in the hands of one who is willing to make a special study of its technique. This method should be reserved for patients with the severest forms of angina pectoris and degenerative changes of the coronary arteries. If surgical intervention is to be undertaken in less advanced cases it should be by open resection.

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THE INTERLAMINAL REMOVAL OF PROTRUSIONS OF THE INTERVERTEBRAL DISC AT THE FOURTH AND FIFTH LUMBAR INTERSPACES

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PRIOR to the epochal paper of Barr, Hampton and Mixter, establishing low back pain and sciatica due to protrusions of the intervertebral disc as a clinical entity, these lesions were removed as spinal canal tumors through the classical laminectomy. Familiarity with the surgery of any lesion leads to the simplification of surgical technique and several surgeons found it helpful to do hemilaminectomies in these cases instead of the more extensive laminectomy.

Most of these lesions occur at the fourth or fifth lumbar interspaces and the interlaminal spaces at these levels are larger than elsewhere in the spine. When the interlaminal space is large (Fig. 4), access may be had to the anterolateral aspect of the spinal canal simply by dissecting out the ligamentum flavum without the removal of bone (2). If the interlaminal space is smaller (Fig. 6a) it frequently is possible to remove the disc protrusion by enlarging the opening by nibbling away the edges of the adjoining laminas (Fig. 6b). This can be done most easily with a small punch-like rongeur that bites at a right angle to its long axis, such as the Ferris-Smith modification of the Kerrison rongeur forcep (3). In cases in which the laminas are further from the surface than usual and in which the interlaminal space is narrow (Fig. 5a) it may be necessary to remove adjoining portions of the spinous processes and laminas (Fig. 5b) to have access to the lesion.

For lesions at the fifth lumbar interspace, it is our practice to make a vertical skin incision from the fourth lumbar spinous process to a point just below that of the first sacral. The interspinous ligament is split between the fifth lumbar and first sacral spinous processes, the muscle is reflected with the periosteum from the spinous processes and laminas outward to the articulation and is held with a Hibbs retractor. The ligamentum flavum is dissected with a small periosteal elevator from the edges of the laminas and is removed with a pituitary rongeur. This exposes the dura, the nerve root, and the lesion which can be seen or palpated with a blunt in-

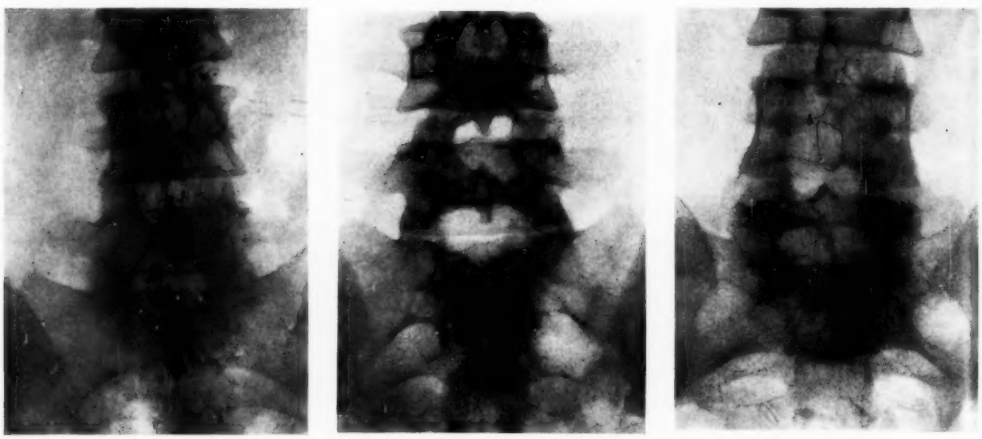

Fig. 1. Postoperative roentgenogram of a spine from which a disc protrusion was extracted without bone removal.

Fig. 2. Postoperative roentgenogram showing bone defect (arrows) resulting from the interlaminal removal of a disc protrusion at the fourth lumbar interspace.

Fig. 3. Postoperative roentgenogram showing defect in lamina (arrows) and spinous process (dotted line) necessary for removal of a protruded intervertebral disc.

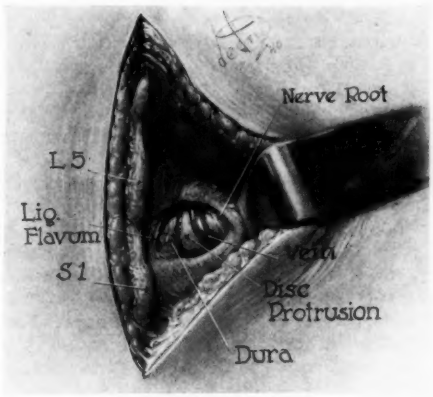

Fig. 4. A case in which removal of a disc protrusion is possible without the removal of bone.

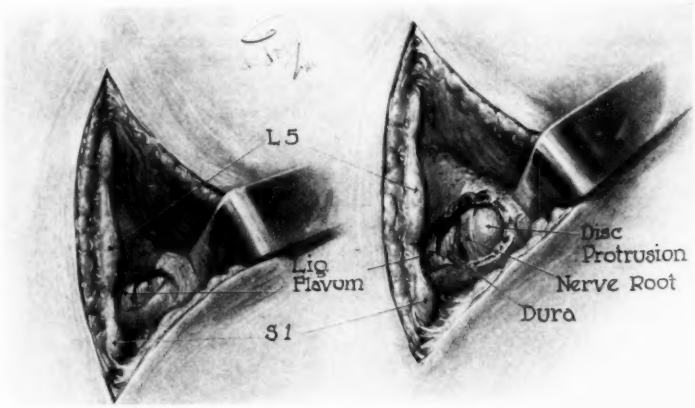

Fig. 5. A laminal variant requiring a minimal removal of bone to allow extraction of a disc protrusion.

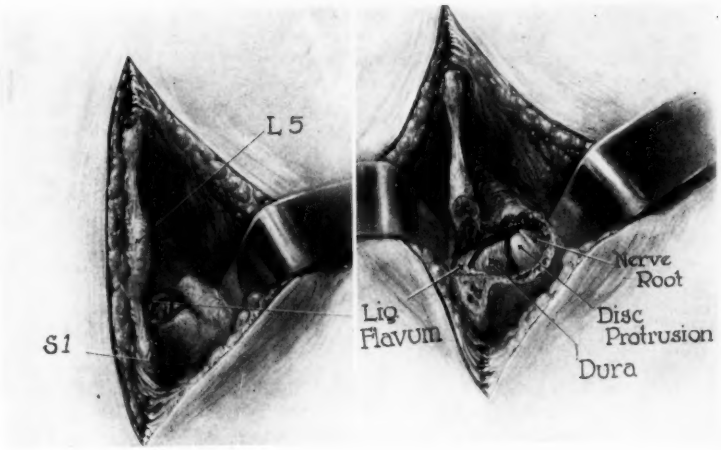

Fig. 6. Closely set massive spinous processes and laminas require the removal of more bone to allow access to the protruded disc.

strument, on the anterior surface of the canal. If necessary, the edges of the laminas are nibbled away with the punch sufficiently to allow the nerve root to be retracted in the direction of least resistance. The dome of the mass, which still may be covered by the posterior spinal ligament, is incised and the mass is teased out with the pituitary rongeur. If lipiodol had been used for diagnosis, the dura and arachnoid are incised, and the table is tilted head upward to allow the oil to drain out. The small incision is closed with one or two silk sutures on small curved needles. After perfect hemostasis has been obtained, the retractor is removed and the wound is closed in layers with interrupted silk sutures.

In most cases of protrusion of the intervertebral disc, these exposures are adequate, time saving, and not difficult. They conserve bone and since a minimal amount of tissue is traumatized, the wound heals more quickly than in the case of laminectomy. If the lesion should prove to be not removable through so small an opening, the operation quickly can be converted into a hemilaminectomy or a laminectomy. At other levels in the spinal canal where the interlaminal spaces are not so large, more extensive removal of bone usually is necessary.

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CANCER OF THE FLOOR OF THE MOUTH

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CANCER of the floor of the mouth is one of the more malignant but less frequent growths of the oral cavity. It is closely allied but not identical to cancer of the tongue, with which it is sometimes classified in the medical literature. In general, cancer of the floor of the mouth is unsuited to surgical removal and, when treated by radiation, may result in serious complications. Before the advent of modern radiation therapy, these difficulties caused this disease to bear an undeserved reputation for poor prognosis or even incurability. The present report is based upon an unselected and consecutive group of 103 cases of cancer of the floor of the mouth including all-comers in all stages of the disease who applied to the Memorial Hospital between the years 1930 and 1934, inclusive.

DEFINITION

In the present report, the term "cancer of the floor of the mouth" designates those growths arising in that crescent-shaped area which lies between the inner surface of the lower gum and the under surface of the tongue. In the literature this term has the sanction of long usage, although some writers include this group of tumors with "lingual cancer." Others separate "sublingual" or "infralingual" from "lingual" cancer, but do not specify a line of demarcation between the two. In some of these reports it is apparent that the term "sublingual" cancer refers both to growths of the floor of the mouth and to those of the undersurface of the tongue. From the practical standpoint, there is little advantage in grouping all tumors in these areas under one heading, and since each possesses distinct anatomical and clinical features and therapeutic indications, it is undoubtedly best to designate a specific group known as "cancer of the floor of the mouth," as described anatomically here.[1]

From the Memorial Hospital.

[1] *Anatomy of the floor of the mouth.* Anatomists do not clearly define the limits of the floor of the mouth, although most of them at least mention the structure. By definition, the tongue consists mainly of a muscular body covered by mucous membrane. On its under surface, this mucous membrane is closely applied to the genioglossus and hyoglossus muscles and is then reflected from the surface of these muscles to pass over the *floor of the mouth* onto the inner surface of the lower gum anteriorly and laterally. The floor of the mouth, therefore, is a crescent-shaped area bounded laterally and anteriorly by the inner surface of the lower alveolus, mesially by the reflection of the mucous membrane onto the muscular body of the tongue, and ending posteriorly about on a line between the third molar tooth and the foliate papillæ of the tongue (Fig. 1).

Beneath the floor of the mouth the submucosal space between the under surface of the tongue and the mandible contains loose areolar tissue in which lie several structures. The sublingual salivary glands lie immediately beneath the mucous membrane laterally, one on each side, opening by 10 to 12 ducts along the longitudinal ridge known as the plica sublingualis. The submaxillary salivary ducts run forward mesial to the plica sublingualis to open near the midline, one on each side of the frenum. Posteriorly deep in the floor of the mouth there is a short forward projection of the submaxillary salivary gland which rests on the posterior edge of the mylohyoid muscle. These deep structures lie embedded in loose areolar tissue and are supported from below by the mylohyoid muscle laterally and the genioglossus anteriorly.

Anatomy of the lymphatic drainage of the floor of the mouth. From a consideration of the topography of the metastatic nodes which developed in the present series, it seems probable that the nodes which serve as the first filter for the floor of the mouth are the preglandular and intracapsular nodes of the submaxillary group. The prevascular and retrovascular nodes in the upper posterior part of the submaxillary triangle are less often involved first than the aforementioned group. Contrary to what might be expected, the submental nodes do not lie in the main drainage path from the floor of the mouth. Obviously, there are bypasses into the upper deep and occasionally into the middle deep cervical groups of the jugular chain, since metastases often appear first in these areas. In only one case in our series were the submental nodes involved first, and it is hardly reasonable to believe that most submental nodes in cancer of the floor of the mouth occur by retrograde lymph flow from the submaxillary group.

ETIOLOGY

Incidence. At the Memorial Hospital cancer of the floor of the mouth comprises 17 per cent of all intraoral tumors, 7 per cent of all cancer of the upper respiratory and alimentary tracts, and 1.43 per cent of all admissions for cancer. It is about half as frequent as cancer of the tongue. Ducuing reports 40 cases (0.42 per cent) in 9,504 cancer admissions to the Anti-Cancer Center in Toulouse, France. Fraser states that these growths make up 10 per cent of all intraoral cancer, and Stout found that 5 per cent of his series of intraoral cancer originated in this area. There are few reports in the literature on the relative incidence of cancer of the floor of the mouth, which many clinics apparently do not recognize as a separate anatomical entity but rather as a subgroup of cancer of the tongue or lower jaw. So far as we can learn, the term is not used in the average mortality statistics.

Age. In the series reported here, the average age was about 61 years, which is slightly higher than in lingual cancer (59 years) or intraoral cancer in general (about 57 years). Because the series is small, however, this slight variation may be only accidental. Schinz and Zuppinger found an average age of 55 years. The oldest patient in the present series was 89, the youngest 36. Only 10 per cent of the patients were under the age of 50, and over half were past 60. We have found practically no other specific references to age in the literature.

Sex. Cancer of the floor of the mouth appears to be more exclusively a disease of the male (98 per cent in our series) than does any other single

Fig. 1. Anatomy of the floor of the mouth. The floor of the mouth is a crescent-shaped area bounded laterally and anteriorly by the inner surface of the lower gum, mesially by the reflection of the mucous membrane onto the muscular body of the tongue, and ending posteriorly about on a line between the third molar tooth and the foliate papillæ of the tongue.

anatomical form of cancer which can develop in both sexes. Heretofore, cancer of the lip (95 per cent in males) has commonly been regarded as the best example of unique male susceptibility. In the closely allied cancer of the tongue, only 85 per cent of the cases occur in males. Schinz and Zuppinger report a 99 per cent male incidence in cancer of the floor of the mouth, Golden 98 per cent, and Ducuing found no females in his forty cases.

Position of the growth. Cancer of the floor of the mouth arises most frequently in the anterior portion to one side of the midline in close proximity to the opening of the submaxillary ducts. Although these growths seldom originate in the midline (3 per cent), they eventually cross the midline by direct extension in almost half of the cases. In the present series, few lesions developed in the middle third of the area. In about 12 per cent, the tumors originated posteriorly opposite the second or third molar.

Causative factors. Chronic irritation as a predisposing factor is not so easily demonstrated in malignant tumors of the floor of the mouth as in cancer of the tongue or of the mucosa of the cheek. In the latter two anatomical sites, evidence of chronic glossitis, atrophy of the papillæ, or leucoplacia is found in a higher proportion of cases. From the anatomical standpoint, it is obvious that the floor of the mouth is protected to a considerable degree from dental trauma and from irritating contact with food and drink. Therefore, chronic irritation in the floor of the mouth is most likely to be a part of generalized glossitis and stomatitis. Theoretically, at least, it is possible that some irritation in this dependent area can be caused by retention and fermentation of oral secretions and food particles. In examining the floor of the mouth, it is not uncommon to find 1 or 2 cubic centimeters of cloudy saliva containing débris of food and other foreign material.

An analysis of the best known forms of chronic irritation of the oral mucous membrane, which occurred in the present series, will be discussed separately.

Tobacco. According to the case histories, 80 per cent of the patients admitted the use of tobacco, usually smoking. In attempting to assay the importance of this factor, one meets the same difficulty as in other forms of intraoral cancer; that is, about the same percentage of normal adults are addicted. So far as this series is concerned, there is little evidence that cancer of the floor of the mouth arises frequently as the result of an excess of the tobacco habit.

Chronic trauma from ill-fitting dentures. It has been emphasized frequently that in many forms of intraoral cancer, ill-fitting dentures are a responsible etiological factor, a theory which is usually difficult to demonstrate in individual cases. It is obvious that when cancer arises in the oral cavity the patient, searching for some reasonable explanation, often ascribes the lesion to irritation from a denture which may have become ill-fitting because of the growth rather than vice versa. Such a tendency to ascribe cancerigenic capacities to alleged local trauma is common among both the laity and the medical profession in all forms of cancer. The fact that one event may appear to follow another is not always proof that the first is the cause and the second the effect. It will be found that most dentures become loose and ill-fitting after 4 or 5 years of use and that the average person continues to wear his dental plates for 15 to 20 years or even longer without change. If there were a frequent etiological connection, cancer of the floor of the mouth and of the gums should be much more frequent.

In this series, 29 per cent of the patients were completely edentulous, a not uncommon percentage in patients averaging 60 years of age, and in only 3 cases (3 per cent of the total group) did the patients believe that a denture had caused irritation in the floor of the mouth.

Syphilis. In the present series, Wassermann tests were made in 69 cases and were positive in only 7 (10 per cent). This percentage of syphilis

is only about one-third that found in tongue cancer, but is nevertheless greater than the average incidence of syphilis in males of the corresponding age group—6 per cent—(26). It is probable, therefore, that syphilis plays a definite though minor rôle in the etiology of cancer of the floor of the mouth.

Leucoplacia. Leucoplacia, when widespread and generalized throughout the oral cavity, is frequently observed in the floor of the mouth, and was specifically noted in some portion of the oral cavity in 26 (25 per cent) of the present series. This incidence is less than that found in cancer of the tongue (46 per cent) and of the cheek (70 per cent). These findings indicate that chronic irritation is probably a less prominent etiological factor in the floor of the mouth than in other forms of intraoral cancer.

HISTOPATHOLOGY

In the floor of the mouth as in most parts of the oral cavity proper, the most frequent histological type of cancer is epidermoid carcinoma grade II (75 per cent) with grades I and III much less common. Adenocarcinoma arising in minor salivary glands occasionally occurs (3 per cent). The radiosensitivity of the floor of the mouth cancer is higher than that of the anterior portion of the tongue or the mucosa of the cheeks and resembles that of the base of the tongue and pharynx. As shown in a previous report (18), there is no adequate explanation of this fact from a histological standpoint.

SYMPTOMS, MORBID ANATOMY, AND CLINICAL COURSE

As in many other intraoral tumors, the most common first symptom in this disease is the patient's discovery of the lesion itself. During mastication, the tip of the tongue is frequently inserted into the floor of the mouth. The tactile sense of this portion of the tongue is very acute, and it is obvious that no irregularity of the oral mucous membrane which can be reached by this structure will long escape discovery. In 10 of the present cases the first symptom noted by the patient was the appearance of a cervical node, and in these the primary lesion was discovered first by the examining physician at Memorial Hospital. The primary lesion in the latter cases averaged 2.25 centimeters in diameter and was usually situated in the anterior one-third of the floor of the mouth.

The symptom next in order of frequency is slight pain or tenderness, which is usually associated with infection of the growth and, therefore, seldom occurs in the earlier lesions. When pain or tenderness is given as the first symptom, the lesion is usually fairly well advanced, and upon closer questioning the patient will, as a rule, admit that he has been aware previously of an irregularity in the floor of the mouth.

The earliest lesions in this series consisted of superficial deep red, slightly ulcerated plaques about 5 millimeters in diameter, infiltrating the mucous membrane. In all of these early lesions the complaint was of "soreness" in the floor of the mouth, and the patients were of the apprehensive intelligent type. It is probable, however, that only a small percentage of the growths begin in this manner, since in the present series the average diameter of the growth on admission was a little over 2.5 centimeters and the average duration of symptoms was about 7 months, indicating that pain or tenderness had neither been an early nor a marked symptom.

When the growth reaches a size of about 1 centimeter, it tends to fungate out from the mucous membrane, to form an irregular, nodular, sometimes slightly pedunculated, ulcerated tumor which may spread either posteriorly onto the undersurface of the tongue or anteriolaterally onto the inner surface of the alveolus (Fig. 2). In other instances, the lesion may deeply invade the floor of the mouth, to penetrate the subcutaneous tissue in the submental region, sometimes simulating metastases to the submental lymph nodes (Fig. 3). Such deep invasion is usually accompanied by obstruction to the submaxillary duct, so that there is associated swelling and tenderness of the submaxillary gland on the involved side (Fig. 4). Invasion of the sublingual salivary gland appears to cause no specific symptoms. In advanced cases, there is usually extension into all of these structures. The tongue, when deeply invaded, becomes fixed; surface necrosis occurs with sepsis; and pain and salivation due to the inability to swallow become prominent symptoms.

With deep invasion of the tongue and floor of the mouth, the growth becomes fissured with localized cellulitis. Surface necrosis may develop, accompanied by pain on movement of the tongue or on swallowing. Dysphagia results in profuse salivation. With deep erosion, hemorrhage frequently occurs from the lingual arteries, and the trauma incident to local tamponage in an effort to control the hemorrhage results in increased necrosis and extension of the sepsis. These complications combine to produce malnutrition and cachexia, and when the disease is uncontrolled locally, death occurs in most cases before the cervical or visceral metastases become prominent.

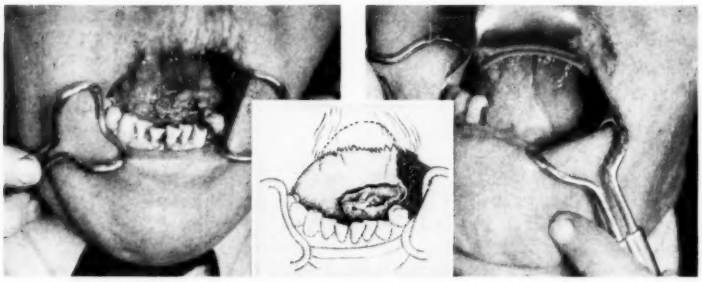

Fig. 2. Patients with cancer of the floor of the mouth are fortunate if they happen to be edentulous at the beginning. The absence of teeth facilitates the accurate centering of the peroral cylinder for x-radiation of the floor of the mouth. The laceration of the gums following extraction of the teeth markedly increases the danger of such later complications as osteomyelitis. At left, cancer of the floor of the mouth of 2 months' duration. Diagrammatic insert shows extent of lesion. The healed condition after treatment by a combination of x-radiation and radon seeds is shown at right. Patient has remained well for 2 years.

Metastases. Although cancer of the floor of the mouth is considered by all writers on this subject to be a highly malignant form of oral cancer, a statistical analysis of the metastases in the present series does not indicate that the malignancy of this disease depends upon an early or unusual capacity to metastasize. In the present series, 35 (34 per cent) had metastases on admission, which, although more than the figure in a recently analyzed series of cancer of the lip (24 per cent) is a little less than in cancer of the tongue (39 per cent). After admission, an additional 24 per cent developed metastases, so that a total of 58 per cent had metastases at some time during the course of the disease, which is about the same as in cancer of the tongue (62 per cent).

In the present floor of the mouth series, the first lymph node involved was in the submaxillary group (about 66 per cent of those cases which metastasized). In 16 per cent the first node involved was the upper deep cervical group of the jugular chain. Contrary to what one might expect, the submental lymph nodes are not in the direct path of the main lymphatic drainage from the floor of the mouth, and in the present series they were involved first in only one case. As previously mentioned, with deep extension the growth may invade the subcutaneous tissue in the submental region, simulating metastasis to this area. After initial involvement of the submaxillary lymph nodes, the disease in most cases extends to the jugular chain and progresses downward to involve the supraclavicular nodes. In one case in the present series, the first node involved was in the middle of the jugular chain.

In the autopsy records at the Memorial Hospital there are 12 cases of cancer of the floor of the mouth, and in only 1 were visceral metastases

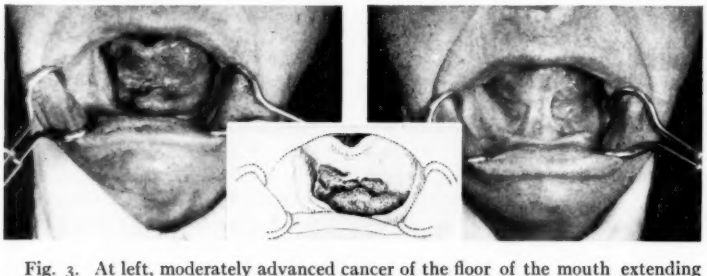

Fig. 3. At left, moderately advanced cancer of the floor of the mouth extending to, but not beyond, the midline. Diagrammatic insert shows extent of lesion. The presence of teeth in the lower jaw renders treatment more difficult. In the average case, it is better to extract them before treatment is begun. At right, healed condition after treatment by peroral x-radiation and radon seeds. Patient has remained well for 1 year.

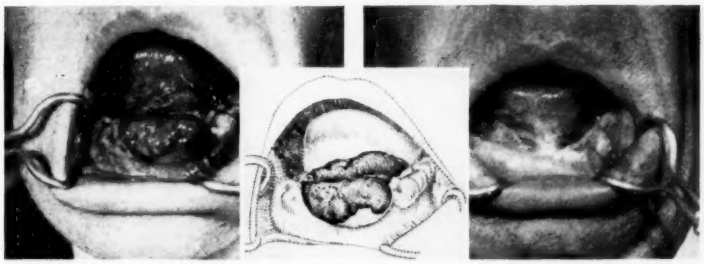

Fig. 4. At left, advanced cancer of the floor of the mouth involving the adjacent gum margin. Diagrammatic insert shows extent of lesion. At right, healed condition following treatment by x-radiation and radon seeds. Patient has remained free of disease for 7 years.

found—liver and lungs. This finding is based on too small a number of cases to be considered conclusive and it may be only coincidental, since at the Memorial Hospital visceral metastases have been found in 29 per cent of patients dead of cancer of the lip and in 40 per cent of those dead of cancer of the tongue. There is no logical explanation for this discrepancy. Dorrance, reporting on both floor of the mouth and lingual cancer, states that in 15 autopsies he found no visceral metastases.

DIAGNOSIS

As in all forms of cancer, a biopsy is essential in the diagnosis of suspected malignant tumors of the floor of the mouth. Furthermore, histological examination is essential as a matter of record in any series subjected to statistical analysis.

Delay in diagnosis. In the present series there was an average delay of 5 months between the first symptom and the establishment of a definite diagnosis. The significance of delay in diagnosis is shown by comparison with other forms of intraoral cancer recently analyzed at Memorial Hospital (17, 19). In a series of cancer of the tongue the interval was about the same, indicating that the severity of the symptoms and the progression of these two diseases is probably about the same. In cancer of the lip there was an interval of about 15 months between the first symptom and admission for treatment, indicating a much more benign and slower clinical course. In few of the cases of cancer of the floor of the mouth was the delay due to procrastination by the medical or dental profession. Three patients were treated by dentists and had teeth extracted under a diagnosis of dental irritation. Three patients had surgical or radiological treatment in other clinics for cervical metastases without discovery of the primary lesion. Two patients had been reassured by a physician that the disease was inflammatory and were told to use antiseptics. In one instance antiluetic treatment had been given over a period of several months under a diagnosis of syphilis, because of a positive Wassermann test.

Differential diagnosis. The differential diagnosis of cancer of the floor of the mouth should not be difficult, since benign ulcerated lesions are extremely rare in this area. A calculus in the submaxillary salivary tract, although producing an indurated palpable mass, should be readily differentiated. The so called "ranula," which is a dilatation of a minor salivary gland and not, as is commonly supposed, a distention due to blockage of the submaxillary salivary duct, should never be confused with cancer. Single herpetic lesions may occur in the floor of the mouth, but may be easily differentiated from cancer by a history of sudden onset with pain and tenderness and the characteristic punched-out appearance of the ulcer. If associated with herpes elsewhere in the oral mucous membrane, the diagnosis is simple. Such chronic and asymptomatic lesions as leucoplacia, hemangioma, and xanthoma should be diagnosed without difficulty.

In dietary deficiencies, especially those due to avitaminosis B, the floor of the mouth is often the site of a subacute inflammation, deep red and sharply delimited in character, which may closely resemble early cancer. In this condition there will usually be an associated mucositis of the tongue as well as radial fissures in the skin at the labial commissures, which indicate the true nature of the disturbance.

GENERAL PRINCIPLES IN TREATMENT OF CANCER OF THE FLOOR OF THE MOUTH

Only early growths of the floor of the mouth not much over 1 centimeter in diameter can be removed surgically even by extensive and locally

Fig. 5. Peroral x-radiation for cancer of the floor of the mouth. With the mouth opened as widely as possible, the floor of the mouth can be exposed for peroral x-radiation by the introduction of a cylinder 2.5 to 4 centimeters in diameter. The large cylinders can be used only in the absence of the anterior teeth in the upper and lower jaws. The extraction of even sound teeth is justified, when necessary to facilitate such procedures.

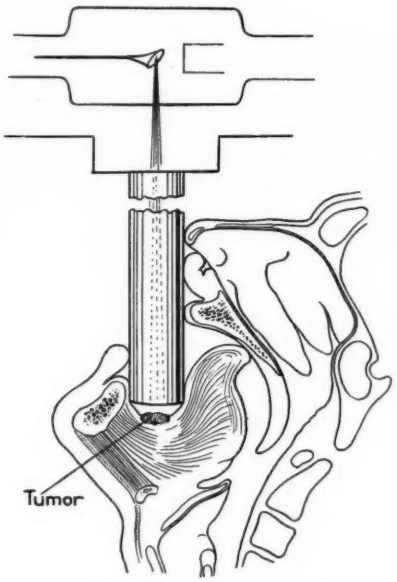

Fig. 5a. Diagram showing application of apparatus.

deforming operations. The larger lesions (the average diameter on admission is a little over 2.5 centimeters) are inoperable from the practical standpoint except by resection of the anterior parts of both the tongue and the mandible. The inoperability of the average case of cancer of the floor of the mouth is admitted by most writers. Among 12 authors who definitely express opinions on this subject, only McKillop and Morestin recommend surgery, and the latter wrote before the advent of modern radiation therapy. Most other authors (2, 6, 11, 12, 14, 22) recommend radiation or a combination of radiation and surgery, with a small number (1, 3, 6, 24) expressing a preference for the cautery or electrocoagulation. Even in the smaller growths, surgical excision necessitates stripping the soft tissues, including periosteum, from the inner surface of the mandible and the removal of the mucous membrane from the under surface of the tongue. Healing of such a wound necessitates sequestration of the exposed cortex of the mandible, and the wound can contract only by pulling the tip of the tongue down

a b

c

Fig. 6. Periscope. A, Master head with 4 centimeter peroral cylinder attached; b, electrically lighted periscope with rheostat; c, physician checking centering of peroral portal by electrically lighted periscope before beginning the irradiation.

into the floor of the mouth, where it becomes fixed to the mandible and practically immobile. If surgical removal were the only form of treatment available, and if it could be successfully applied in a fair percentage of cases, such postoperative defects and deformities would certainly be justified in so malignant a disease as cancer of the floor of the mouth. It must be admitted, however, that a relatively small percentage of cases are operable when first seen, that radiation therapy (either curative or palliative) is applicable in all cases, and that properly applied modern radiation therapy in all comers will produce a higher cure rate than surgery used only in the early and operable cases. It is significant that those authors who recommend surgical excision or endothermy as being superior to radiation therapy make no report on 5-year cures (McKillop, Bloodgood).

Radiation therapy of cancer of the floor of the mouth was formerly given either by interstitial implantation alone or externally through portals directed horizontally through the rami of the mandible and vertically, from below, through the submental region. Such treatment was ordinarily supplemented by interstitial radon. Following radiation under such plans, the incidence of localized radionecrosis of soft tissue and bone was comparatively high. A more recent development is the use of peroral x-radiation (16) given through the open mouth with cylinders centered directly over the lesion itself. When supplemented by radon seeds, the intense radiation dose may thereby be confined to the growth and the immediately adjacent tissues, sparing the outlying normal tissue (especially the mandible), and the incidence of untoward sequelæ can be markedly reduced. There can be little question but that radiation given by the technique described below is the best so far developed in the treatment of cancer of the floor of the mouth.

General hygienic measures. Cancer of the floor of the mouth is commonly associated with a marked degree of local infection, especially when the lesion is deeply fissured, and in any case sepsis usually occurs as a complication of treatment. If teeth are present, they always interfere with treatment and lead to dental complications following radiation. The patient with cancer of the floor of the mouth is indeed fortunate if he happens to be edentulous at the beginning. Both upper and lower teeth anteriorly interfere with the proper insertion and direction of the peroral cylinder, and it is usually necessary to extract or cut them off before treatment. Even if the teeth are perfectly sound, such measures are justified. Healthy teeth may be cut off at the gum level, and the root canals filled so that later dental restorations may be made. There are other reasons for cutting off the teeth rather than extracting them, as will be discussed further under complications. In all cases before beginning treatment, all the teeth should be thoroughly cleaned by a dentist and the patient instructed in the proper use of a tooth brush.

During radiation treatment and until healing is complete, the patient should be instructed to irrigate the mouth with large quantities of warm alkaline-saline solution (1 quart of water with 1 dram each of sodium bicarbonate and sodium chloride). Since the local area becomes tender and painful during the radiation reaction, the taking of food is usually uncomfortable and even painful. The patients must therefore be instructed in the form and nature of a satisfactory diet. In many cases, it must be liquid or at least soft, and directions should be given for the preparation of a properly balanced diet. During recent years it has become more and more evident in the clinic at the Memorial Hospital that a large percentage of intraoral cancers, especially those of the tongue and floor of the mouth, are associated with a chronic glossitis, usually related to some dietary deficiency or avitaminosis. If the diet becomes further restricted due to local discomfort, this dietary deficiency is accentuated. We have found that the local condition and the maintenance of the patient's general health during treatment and recovery is greatly favored by the administration of large quantities of vitamins, the most necessary being the B complex and, though of lesser importance, vitamin C. In the most marked dietary deficiencies, liver is required in the form of liver extract powder in doses of at least 1 teaspoonful, three times a day, dissolved in about 8 ounces of hot water and taken as a bouillon. In lesser degrees of deficiency or during convalescence, this therapy may be given in the form of dried yeast, one heaping tablespoonful three times a day. If the patient cannot tolerate either liver extract or yeast by mouth, or if either is found to be difficult to administer because of local tenderness, then extracts of these substances can be given intramuscularly.

In our clinic all patients attend what is known as the "Clean-Up Clinic" daily during the administration of the fractionated x-radiation and until the reaction has completely healed. At this time the local area is cleansed with a power spray of an alkaline solution or Dakin's solution, and any other deficiency in the patient's hygienic or dietary regimen is corrected.

TREATMENT OF THE PRIMARY LESION IN CANCER OF THE FLOOR OF THE MOUTH

The treatment of cancer of the floor of the mouth by radiation is so far superior to surgical removal that the latter does not merit any detailed discussion. Most surgeons strip the periosteum from the adjacent inner cortex of the mandible, widely and deeply excise the primary lesion and the adjacent mucous membrane and muscle of the under surface of the tongue. The wounds are left open to heal by granulation. Cautery or endothermy methods alone are probably even less desirable forms of therapy in this area. Radiation therapy is undoubtedly best given in the form of fractionated peroral x-radiation through rather narrow portals, supplemented in most instances by radon seeds implanted directly into the growth.

Technique of treatment by peroral x-radiation. In deciding upon the exact factors for peroral x-radiation in this area, the mouth should be opened as widely as possible and the growth visualized through sample cylinders of various diameters. The size, shape, and exact direction of the proper cylinder should be selected so as to expose the primary lesion adequately at an angle of approximately 90 degrees to the incident beam. The diameter of the portal depends to a large extent upon the size of the primary lesion. Only in the smaller growths, with a diameter of 1 centimeter or less, should portals as small as 2.5 centimeters be used, and these can be employed safely only if special attention is given to accurate centering. If there is any question in this regard, the portal had best be at least 3 centimeters or larger in diameter, but it is seldom feasible to give peroral x-radiation to the floor of the mouth through portals larger than 4 centimeters. The detailed technique of peroral x-radiation has been described in a previous report (16).

The patient may be seated in a chair with a head rest, or he may lie on a table with the head propped up by pillows as in Figure 5. The cylinders should be inserted so as to hold the tip of the tongue folded upward and backward. The accurate centering of the peroral cylinders is best checked by a lighted periscope device as illustrated in Figure 6. Since most of these lesions are at the surface, a target skin distance of 35 centimeters or even less is to be preferred, with the other physical factors about as follows: 200 kilovolts, 15 to 30 milliamperes, filter 0.5 millimeter copper plus 1 millimeter aluminum. The dosage must vary with the size of the portal. Treatment may be given either daily or, as a matter of convenience—to patients who live at a considerable distance—every other day or three times a week. The dose of the individual treatment should be adjusted so as to deliver a total of 5,000 to 7,000 r in 20 days with portals 4 to 2.5 centimeters in diameter. In the average case the treatment should be outlined so as to deliver a specified total dose in a period of about 3 weeks, after the completion of which the decision as to the necessity for supplementary treatment by either additional peroral x-radiation or radon seeds must be made empirically on the basis of the intensity of the local reaction and the degree of regression of the primary lesion.

Technique of supplementary treatment by radon seeds. During the course or immediately following the completion of fractionated x-radiation, radon seeds in small, sometimes divided, doses can usually be employed to advantage in the floor of the mouth. The dosage is empiric and can be learned only by experience. The strength of the individual seeds should be small (1 to 1.5 millicuries) and they should be placed, if possible, over 1 centimeter apart. The total dose need seldom be over 5 to 6 millicuries in the average case and can often be cut down to 2 to 3 millicuries. In most clinics the treatment of cancer of the floor of the mouth was formerly carried out almost entirely by the interstitial implantation of radon seeds or needles, and even today such therapy is recommended by some authors. This method is attended by an unnecessarily high incidence of local radionecrosis and osteomyelitis, complications which are particularly serious in the floor of the mouth. By a combination of peroral x-radiation and radon seeds, the frequency of these complications can be greatly reduced, and it is our opinion that interstitial radon alone should now be discarded as a method for the treatment of cancer in this location.

TREATMENT OF METASTASES IN CANCER OF THE FLOOR OF THE MOUTH

The problem in the treatment of cervical metastases in cancer of the floor of the mouth resembles that in cancer of the tongue. If all cases are considered, it will be found that neck dissection is not suitable in the majority, the main reason being that if the growth in the floor of the mouth is extensive, requiring a long convalescence after treatment, or if metastases are present on admission even with a small primary lesion, neck dissection cannot be carried out with safety in conjunction with aggressive treatment to the primary lesion. One solution of this difficulty is the treatment of the metastases as well as the primary by radiation. The choice between neck dissection and radiation for the treatment of late

metastases—those which appear clinically after healing of the primary lesion—is optional and may be made on the basis of the available equipment and the experience of the surgeon with the various methods. In the average favorable case of metastases following complete healing of the primary lesion, it is probable that radiation is at least as successful as neck dissection.

Radiation treatment of metastases. If the nodes occur in the submental or suprahyoid regions, it must be realized that aggressive treatment by irradiation of both the primary lesion and the metastases is apt to be more than the local tissues can tolerate. Deep necrosis with perforation from the floor of the mouth through the submental region is therefore not an infrequent complication, especially if the inner surface of the mandibular arch has been exposed so as to produce osteomyelitis and sequestration of bone in this region. A partial solution of such a difficulty is the use of accurately centered small portals and accurately placed radon seeds both in the primary lesion and the cervical nodes. So far as the cervical nodes are concerned, this end is best accomplished by x-radiation given through small portals limited to the region of the node itself with a margin of not more than 1.5 to 2 centimeters giving total doses of 5,000 to 7,000 r in fractions, supplemented by small doses of radon seeds as in the treatment of the primary lesion. Such treatment can be given to each of several widely separated nodes. The exact technique as used at the Memorial Hospital includes (a) histological proof of the malignant character of the node by aspiration biopsy, (b) the accurate marking of the skin by tattooing directly over the center of the node, (c) the selection of the proper sized portals (3 to 5 centimeters in diameter, and (d) supplementary radon seeds, often in fractionated doses. Treatment by fractionated x-radiation is given either daily or three times a week, and the individual dose so selected as to reach a total dose of 6,000 to 8,000 r in a period of 3 weeks, after which a dose of 5 to 6 skin erythema doses seeds is implanted either through the skin or directly into the node after surgical exposure.

In the present series, 6 cases of cervical metastases proved by aspiration biopsy have remained free of recurrence 5 years following treatment by radiation. There are 3 additional successful cases in which, unfortunately, the histological confirmation of the clinically positive nodes was omitted. All cervical metastases not treated surgically, including the hopelessly advanced, were treated by radiation, and therefore calculations as to the percentage of successes by this method could not be fairly compared to the early and favorable cases treated by surgery.

Neck dissection. Neck dissection in cancer of the floor of the mouth is of limited value. Since the primary lesion usually crosses the midline, either or both sides of the neck may be involved. When metastases occur, they do not long remain directly in the submaxillary or submental regions. A bilateral dissection of these areas is not sufficient, and routine block dissection of both sides of the neck is obviously injudicious. From the practical standpoint, neck dissection should be considered only in those isolated cases in which metastases first appear after complete healing of the primary lesion. Neck dissection when done in these cases should extend from the lower border of the mandible and include the whole cervical region from the midline to the anterior border of the trapezius down to the clavicle. In the submental area the dissection should be wider to the opposite side than in cancer of the lateral border of the tongue, and therefore the operation must obviously be a great deal more extensive than for metastases of the lip.

In the present series, 8 patients with cervical metastases were treated by neck dissection, of which all but one showed histologically positive nodes. Of the histologically positive group, 4, or 57 per cent, remained free of disease for 5 years or more.

COMPLICATIONS

Pain. In the absence of deep invasion with radionecrosis, the pain incident to the treatment of floor of the mouth cancer can usually be adequately controlled by mild narcotics, such as codeine and acetyl salicylic acid. Proper attention to the local hygiene is of great assistance in this regard. When radionecrosis with osteomyelitis occurs, the local pain is often so severe as to be incapable of satisfactory relief by either local cleansing measures or narcotics. This area is supplied entirely by the third divisions of the fifth cranial nerves, and there is no anatomical form of cancer in which neurolysis by alcohol injection is more efficacious. The injections for this purpose should be made externally below the zygoma because, unfortunately, neither the lingual nor the inferior dental nerves can be satisfactorily approached through the mouth for alcoholic injection. The detailed technique for these procedures has been described by several authors (10, 13, 15).

Radionecrosis and sepsis. These complications occur most often when the x-radiation is given through the skin of the chin and submental regions or when interstitial radon is employed in

heavy dosage. Many years ago Quick called attention to the high incidence of radionecrosis when seeds alone were used in the floor of the mouth. Although the incidence and extent of radionecrosis is greatly reduced by the use of peroral rather than external skin portals, this complication in the larger lesions (over 2 centimeters in diameter) cannot be entirely eliminated even by the most carefully and efficiently administered peroral x-radiation and the minimal supplementary dose of seeds.

Due to the dependent position of the lesion in the floor of the mouth, adequate drainage is difficult, and there always remains a pool of saliva and débris in which bacteria may flourish. One of the best means of overcoming this difficulty is frequent irrigation and cleansing of the area by a power spray. The bacterial flora in necrotic lesions of the oral cavity is mainly saprophytic and anaerobic in character and amenable to at least partial control by the application of oxygenic substances. Sodium perborate in solution was formerly used with some success, but a much more efficacious method has recently been developed by Meleney. Zinc peroxide, when activated by autoclaving to 140 degrees for 1 hour and made into a thick suspension to saturate gauze packing, gives off nascent oxygen for periods of 18 to 24 hours. When a necrotic, foul-smelling ulcer so typical of radionecrosis is treated by the application of zinc peroxide on gauze, the odor will usually disappear within an hour or two, and if the slough can be removed, the borders of the lesion assume a pink improved appearance with the complete disappearance of the odor. In most intraoral lesions there appears to be a symbiosis between saprophytes and the ordinary pyogenic organisms. If the anaerobic saprophytes can be eliminated, the normal tissues apparently can soon overcome the pyogens.

Hemorrhage. This complication occurs frequently when there is radionecrosis in the floor of the mouth. The lingual artery enters the tongue on its under surface medial to the hyoglossus muscle and runs forward as the lingua profunda toward the tip of the tongue nearer the under surface than the dorsum. The floor of the mouth is supplied by the branches of the profunda. None of these branches is large enough to be the cause of profuse hemorrhage by itself, but if the necrosis extends deeply and backward toward the under surface of the tongue, the profunda itself may be eroded, and serious hemorrhage may occur. The only immediate method of control is by tamponage, which unfortunately always produces serious trauma and increases the radionecrosis, especially when the tamponage, even though well intentioned, is so energetic as to strip the soft tissues from the inner surface of the mandible. After immediate control by packing, the next step should be ligation of one or both lingual arteries, depending on the extent of the necrotic area. If the radionecrosis crosses the midline, both arteries should be ligated. The surgical exposure for the lingual artery is best accomplished by an incision along the upper anterior border of the sternomastoid muscle, the center of the incision being about 1 centimeter below the angle of the jaw. The anterior edge of the sternomastoid and the jugular vein are retracted posteriorly, and the carotid bulb exposed by blunt dissection. The lingual artery is the first branch of the external carotid to turn upward above the superior thyroid branch, which curves downward. Unless there is very deep backward extension of the necrosis, ligation of the lingual artery alone is sufficient. In order to prevent recanalization, the artery should be divided between ligatures in all cases.

Osteomyelitis. This complication is most likely to occur when unnecessarily large portals have been used for x-radiation and especially when this radiation is given from the outside so as to devitalize the bone of the mandibular arch. When small portals are used perorally and unnecessary radiation to the mandibular arch is avoided and when the dose of radon seeds is kept to the minimum, osteomyelitis is seldom a serious complication. All bone which lies within the zone of a cancer lethal dose of radiation is markedly devitalized by the complete destruction of the finer blood supply. In such bones as the mandible, such an effect is evidenced by a lack of any sanguineous oozing from the cortex or the medulla when the bone is sectioned, although the mandibular artery itself may still be patent. After complete destruction of the intimate circulation, the affected bone may remain *in situ* and function normally *provided that it is not exposed by necrosis or other solution in the continuity of the overlying soft tissues.* If heavily irradiated bone is exposed by any means, osteomyelitis inevitably occurs, and the devitalized bone subsequently acts as a foreign body which must sequestrate and be extruded before healing can occur. It is an error to speak of radio-osteonecrosis of bone as being produced by radiation alone, for even though the bone is completely devitalized it will function just as well as a piece of ivory or a metal plate, *provided that infection is not introduced.*

The most frequent cause of osteomyelitis in cancer of the floor of the mouth is the exposure of

the inner surface of the mandible by necrosis of overlying soft tissues. A second cause is the extraction of teeth so as to expose the heavily irradiated bone in the tooth sockets. Once infection has been introduced and osteomyelitis has occurred in an area of heavily irradiated bone, the infection usually spreads through the entire radiated area of bone, all of which must sequestrate before healing can take place. This fact is one of the best arguments for the use of the smallest feasible portals.

When osteomyelitis occurs in the anterior mandibular arch with necrosis in the floor of the mouth, there is usually an extension through the skin of the submental region. The anterior portion of the mandibular arch finally sequestrates, producing a marked deformity. The normal contour of the chin is lost, and the tongue, having lost its anterior support, recedes backward into the pharynx. The perforation through the submental region can be closed by plastic repair, but articulated speech is greatly altered. Despite the cosmetic and functional deformity, these patients may live on quite comfortably in excellent health. The treatment for osteomyelitis should be conservative, and operative removal of bone should be deferred until the severity of the symptoms warrants it or until it is certain that the further progress of the involvement has ceased. In many instances, it is best to defer the removal of the sequestrum as long as possible or until it has become loosened by itself.

Osteomyelitis occurred in about 25 per cent of the present series. It has become much less frequent in recent years since the use of small peroral portals for x-radiation has been made routine. It occurred more frequently (36 per cent) in the cured cases than in the fatal cases (18 per cent), which indicates that it is justifiable in an effort to control an otherwise fatal disease. We do not concur, however, in the opinion of some authors that osteomyelitis of the jaw is inevitable with adequate radiation treatment.

Dental complications. As has been previously mentioned, the patient with cancer of the floor of the mouth is fortunate if he is edentulous. Dental complications may serve both as a cause and as a result of osteomyelitis. Cancer lethal doses of radiation cannot be given safely over large areas through the gingival margin. Under such dosage, the teeth are devitalized, the gums recede, and serious dental sepsis occurs. Finally the teeth become painful and loose, and if they are extracted the tooth sockets do not respond as in the normal by filling with granulation tissue, so that they may finally heal over with mucous membrane. In the typical case, the devitalized bone remains exposed in the tooth socket and diffuse osteomyelitis soon becomes evident.

The incidence of these complications is greatly reduced by using the smallest portal which will adequately cover the primary lesion. Heavy irradiation of the teeth and the gingival margin cannot be entirely avoided, but in these cases subsequent difficulties may be largely prevented by cutting off the teeth rather than extracting them, killing and extracting the nerves, and filling the root canals, leaving the roots in place permanently, if possible. In some instances, dental restorations can be made.

Adenitis of the submaxillary salivary gland. In cancer of the floor of the mouth, there is practically always invasion of the submaxillary salivary duct with obstruction to the outflow of saliva and, in some cases, the entry of bacteria into the duct and gland. This complication seldom goes on to frank suppuration within the salivary gland itself, but there may be marked swelling and tenderness in these regions. At times the swelling of the submaxillary salivary gland may closely simulate metastases, and the differential diagnosis is important since there is a lymph node which lies deeply embedded within the submaxillary salivary gland, to which metastasis may occur. Although productive of some pain and discomfort, the infection of the submaxillary salivary gland seldom calls for any special measures of treatment except that the gland should always be removed when a neck dissection is performed.

Recurrent and residual cancer. In the present series following treatment by radiation, there was recurrent or residual cancer at the site of the primary lesion in 20 cases, or 19 per cent. Next to metastasis, this complication is one of the most serious in cancer of the floor of the mouth and occurs more often than in cancer of the lip (6 per cent). The frequency with which it occurs may often be due to the surgeon's effort to use the minimal effective dose and to avoid as much as possible the danger of radionecrosis and osteomyelitis. In our series, of the 20 cases of local recurrence, subsequent treatment was successful in 7, or 35 per cent.

PROGNOSIS

Cancer of the floor of the mouth has always been considered one of the most malignant of intraoral tumors. From the reports of many writers, one might infer that the disease is practically incurable. The results in our clinic appear to be about the same as in cancer of the tongue. It is probable that the former bad prognosis of

TABLE I.—FACTORS INFLUENCING THE 5 YEAR CURE RATE IN 87 CASES (DETERMINATE GROUP) 1930 TO 1934, INCLUSIVE

	Total number of cases	Number of 5 year cures	Per cent of 5 year cures
Age in years:			
Below 40	2	2	100
40 to 49	9	3	33
59 to 59	24	9	38
60 and over	51	8	16
Not stated	1	0	0
Position of the growth:			
Anterior one-third	73	20	27
Middle one-third	2	0	0
Posterior one-third	12	2	17
Crosses midline	42	6	14
Histopathology:			
Epidermoid carcinoma grade I	5	2	40
Epidermoid carcinoma grade II	65	16	25
Epidermoid carcinoma grade III	8	3	38
Epidermoid carcinoma ungraded	7	0	0
Adenocarcinoma	2	1	50
Metastases:			
None on admission	59	18	31
Present on admission	28	4	14
Developed after admission	18	6	33
Metastases sometime	46	10	22
None at any time	41	12	29

TABLE II.—5 YEAR END-RESULTS 1930 TO 1934, INCLUSIVE

Total number of cases		103
Indeterminate group		
Dead as a result of other causes without recurrence	13	
Lost track of without recurrence	3	
Total number of indeterminate results		16
Determinate group		
Total number minus those of indeterminate group		87
Failures		
Dead as a result of cancer	60	
Lost track of with disease	5	
Total number of failures in treatment		65
Successful results		
Free from disease after 5 years or more		22
Net 5 year end-results		
Successful results divided by determinate group (22/87)	(per cent)	25

This series consists of all histologically proved cases of cancer of the floor of the mouth, both early and advanced, admitted during the specified period. Only those patients are excluded who, for any reason, were unable to return for treatment, palliation and observation in the out-patient department, and those who were lost track of within the first month after no more than one or two visits (clinic shoppers).

cancer of the floor of the mouth was due mainly to the inadequacy of purely surgical measures to deal with this tumor except in a small percentage of early cases. A statistical analysis of the main factors which influence the prognosis are given in Table I. The most significant of these will be discussed separately.

Age. The average age of the patients on admission was considerably older (5 years) than the average of those who survived 5 years after treatment. An analysis of the 5 year survivals by age decades indicates that the prognosis is less favorable with advancing age—16 per cent after the age of 60 as compared to 25 per cent in the whole group. The total number of cases is so small that the latter figure must not be taken as absolutely representative of the facts in this regard. The poorer prognosis in the older age groups is characteristic of those anatomical forms of intraoral cancer in which complications are serious, as for instance cancer of the floor of the mouth or tongue. In cancer of the lip, in which aggressive treatment introduces relatively few serious complications, the age of the patient does not markedly influence the prognosis.

Position of the growth. The lesions situated anteriorly offer a better prognosis (27 per cent) than those which arise posteriorly (17 per cent). The average diameter of all cases was 2.7 centimeters. The average diameter in the 5 year survivals was about 2.5 centimeters as compared to about 3 centimeters in the fatal cases. The duration of symptoms in the total group was a little over 6.5 months. In the cured group the average duration was 5 months as compared to about 7.5 months in the fatal cases.

Histopathology. In our relatively small group of cases, the histological grade of the tumor apparently did not seriously affect the prognosis. The variations in groups I and II are probably only coincidental.

Metastases. As in all forms of cancer, the presence or absence of clinically demonstrable metastases on admission is one of the most important factors in the prognosis. When present on admission, the 5 year survival rate was only 14 per cent, and the variations in the survival rate depending upon other classifications in this regard are shown in Table I. In those cases in which metastases did not appear at any time during the course of the disease, the cure rate was over 40 per cent.

END-RESULTS

In the present series the net 5 year cure rate (calculated on the determinate group) is 25 per cent (Table I). Of the total of 103 cases, 87 can

be classified as determinate. The 16 remaining are considered indeterminate for the following reasons: 13 died of unrelated causes after freedom from disease for at least a year before a 5 year period had elapsed; 3 were lost track of before the end of 5 years without recurrence for at least a year following treatment. Of the 87 determinate cases, 60 died of disease and 5 were lost track of with disease, a total of 65 failures in treatment.

A survey of the literature reveals few reports of 5 year end-results which include sufficient data so that the figures can be accepted without reserve. Regaud, in 1932, reported 77 cases treated by radiation at the Curie Institute with 22 per cent 5 year cures, but he does not give sufficient data to establish whether this figure represents net or gross cures, nor does he account for those lost track of with and without disease. Berven (2, 3) reports that between the years 1906 and 1930, 40 patients were treated at the Radiumhemmet, of whom 14, or 35 per cent, survived 5 years without recurrence, but he does not state whether all cases in all stages of the disease are included in this group, and he does not account for any patients dying of unrelated causes. Golden reports on 46 patients of whom 12 were lost and only 3 were free of disease for one year. Bloodgood, Dorrance, McKillop, and Cutler simply state that the prognosis is unfavorable and make no specific mention of any cures of their own. Ducuing reports 40 patients treated by radiation in which only one survived for at least 3 years. There are, however, several reports of single cases in which the patient survived more than 5 years.

SUMMARY

This series consists of 103 unselected consecutive cases of all comers with cancer of the floor of the mouth in all stages of the disease, admitted to Memorial Hospital during the years 1930–1934, inclusive. The etiology, clinical course, method of treatment, end-results, and prognosis are described in detail. The net 5 year cure rate is 25 per cent.

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A COMPLETE TECHNIQUE OF HEMORRHOIDECTOMY

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HEMORRHOIDECTOMY is an ancient operation. Galen ligated the base of the internal hemorrhoid on each side and allowed the mass to slough away. Hippocrates was the first to practice excision. Since the original operations, many ways of curing hemorrhoids have been described. A good many methods are not new but are merely repetitions of surgical procedures already devised.

Our method of hemorrhoidectomy justifies a complete, detailed description, because it corrects something that has previously been overlooked. We would call attention to a fourth hemorrhoid, which is almost constantly present and the removal of which has resulted in a much higher percentage of permanent cures than that given by any previous technique. Complete details of our method will be given to enable any first class proctologist to try it. Some of the individual features of the operation have been used previously by other proctologists. However, a careful scrutiny of the literature fails to reveal any single operation which has taken advantage of as large a number of the refined maneuvers included.

INJECTION TREATMENT OF HEMORRHOIDS

There are still some unscientific men who use the injection method, and quite often these men have large rectal clinics. We have watched some of these men at work, and have even read one of their textbooks as well as a volume on their clinics. The fact that men with little skill can use the injection method with such ease, is in itself a condemnation of it, although not an absolute one. Skillful men of the caliber of McPheeters bring the art of injection to its highest point and would never be found guilty of using the method in the presence of infection or of an acutely thrombosed, internal, strangulated, prolapsed hemorrhoid. We contend that proper injection can be done by only a relatively few, expert proctologists.

Hemorrhoids may be classified in three general groups: (1) those with painless bleeding at defecation—in these anemia is often present; (2) those in which there is a spontaneous reduction of prolapse after defecation; and (3) hemorrhoids which prolapse very easily and may occur after any muscular effort such as coughing, dancing, or walking.

Injection is usually recommended only for hemorrhoids of the first degree. Very occasionally it may be used for those of the second degree.

Injection is contra-indicated in many instances: in the presence of pain which is an absolute contra-indication since it usually means infection, such as fissure, ulcer, or abscess; in the presence of cryptitis and papillitis; in the presence of extensive prolapse, often with strangulated or inflamed piles; in the presence of large external skin tabs, which usually mean deeper associated anorectal lesions; in the presence of polyps; and in young patients. Buie and Rankin mention, in some instances, an idiosyncrasy to a drug as a real danger to injection.

In our own experience, which is corroborated by that of the Mayo clinic, it is rare to find internal hemorrhoids in the absence of external ones. Similarly it is nearly as rare to find external ones in the absence of internal ones. This is in direct contradiction to some of the literature, which in many cases states that there are four to seven times as many internal as external hemorrhoids. Unless acute thromboses are present, the surgeon should remove both types of hemorrhoids at the same time. This makes the reasons for injection in most cases, become more and more difficult to understand.

The Mayo clinic proctologists have found uncomplicated hemorrhoids to be productive of little, if any, discomfort and in this we agree. Pain is the symptom of a complication, usually infection, which demands surgical care. It is then that the patients finally reach us seeking relief, and it is time for surgery. Evidently, when hemorrhoids are symptomless and there is not much bleeding, patients do not come to the proctologist.

Once again, we do not deny that to the quack, so called "rectal specialist," the injection treatment is a boon. Unscrupulousness sometimes overcomes better judgment especially if the patient can pay and unfortunately in the hands of such men hemorrhoids are *always* found and the injection treatment instituted. The experience the quacks thus gain, is by the sadder experience of the patients; they learn what and where the pectinate line is and not to inject below it or too close to it. These statements of course do not apply to such men as McPheeters. The relatively

few high class proctologists who are experts with the injection technique will agree in general with its shortcomings, including recurrences.

Briefly summarized, the principal objections to injection treatment of internal hemorrhoids are: (1) the condition is too advanced; (2) the presence of complications, such as thrombosed, prolapsed hemorrhoids; (3) the presence of associated pathological conditions such as infection and external hemorrhoids.

DIFFICULTIES ENCOUNTERED WITH OTHER SURGICAL METHODS

1. Clamp and cautery. The clamp and cautery method is one of the most commonly used. It is not the technique of the proctologist but of the general surgeon who does occasional proctological work. We have many objections to it, most of which have been verified by numerous other proctologists. Not one of the least of these is the failure to remove associated pathological lesions in the anorectal area at the time of the operation.

The Mayo clinic group has stated that deformity is often present about the anal margins, due to the piling up of skin and to the existence of large varicosities beneath the surface of the skin. When the clamp and cautery operation is done, frequently little effort is made to dissect out the varicosities beneath the anal margins before the clamp is applied.

Lockhart-Mummery has said that the clamp and cautery operation is not so complete, and that there is a likelihood of recurrence following its use. Frequent complications are stricture and secondary hemorrhage. Mechling has condemned the searing of the mucous membrane of the anorectal area for these same reasons, as well as for the leaving behind of crushed, burned tissue. Hirschmann has said that the use of a hot iron in a mucus lined cavity is too prone to be followed by strictures. As we well know, there is a mucocutaneous covering over the lower part of the anorectal area, whereas the upper portion is covered only by mucous membrane.

Loring has shown that an effective treatment for prolapse of the rectum is removal of the internal hemorrhoids with elliptical strips of mucosa, by the clamp and cautery procedure. This method relies on the formation of scar tissue which holds up the rectum but causes a consequent diminution in the caliber of the rectal lumen. Therefore a case of prolapse must have an accompanying enlargement of the rectum to be suitable for this type of operation.

2. Ligature operation. The ligature operation has been used by many men, notably Yeomans, Hirschmann, and Goodsall. The danger of recurrence is admitted. Mechling has condemned ligation of the anal membrane for many reasons, such as pain and hemorrhage. We have tried it and have found that the patients suffered too much postoperative pain. Three of our patients had postoperative hemorrhage, and this has been the experience of others. Associated pathological lesions are not removed.

3. The Whitehead operation, and its modification, the amputative method. As Yeomans has said, the Whitehead operation has rightly become almost obsolete. A few men hang to it, tenaciously, possibly because of their actual, exceedingly great skill in surgical technique, and a certain, justifiable degree of pride in exhibiting it.

Mechling has stated that it is never necessary to do an amputative type of hemorrhoidectomy, since healing is never primary and is followed by granulations producing stricture, and prolapse of the mucous membrane constituting the aptly termed "Whitehead deformity." Hirschmann has adequately shown how incontinence with eversion and cutaneous overgrowth has occurred.

We do not use the operation. We can speak only from the experience gained by the sad cases, which have come to us, with functional and anatomical deformities following its use.

4. Condemning oversewing and burying of stumps of mucocutaneous tissue under oversewed mucosa. The burying of crushed mucocutaneous tips or teats of tissue under an oversewed mucosal surface is a violation of good surgical principles, especially when this is done in an infected area. The anorectal region is constantly being flooded with infected material, i.e. feces. This crushed cutaneous tip often undergoes sloughing, and we have tried the method a few times to verify this fact. Any infection buried beneath the oversewed mucosa can become more virulent with ease. Abscesses and strictures take place too frequently.

The Mayo clinic makes use of this maneuver to a certain extent. However, the extreme skill of their operators and the careful after-care of their patients causes a low number of complications. This also applies in the case of Lockhart-Mummery, who uses oversewing of the mucosa.

Location of any postoperative hemorrhage is much more difficult in the presence of oversewed surfaces. This is as true here as in the case of a postoperative hemorrhage within a closed abdomen.

5. Miscellaneous. a. Cutting circularly around the anal orifice. This is against one of the funda-

mental rules of surgery, as it tends to produce a stricture. In the past, Yeomans and Lockhart-Mummery have used modifications of this technique. If the surgery of the stomach and the intestine is recalled, the fallacy of this procedure is readily perceived.

b. Use of local anesthetic—except on thrombotic, external hemorrhoids demanding immediate care. This procedure tends to distort the tissues. Accurate removal of the proper amount of tissue is difficult for us to judge. The Mayo clinic is to be commended for their development and extensive use of caudal and caudal-sacral block anesthesia in the field of anorectal surgery.

c. Immediate removal of internal hemorrhoids in the presence of edema of the rectum, infection and acute thromboses of internal hemorrhoids. The Mayo clinic group has shown that it is difficult to judge the amount of tissue to remove if there is much edema. This is observed when thrombotic, internal, protruding piles are replaced. If infection is present, there is the same difficulty plus the dangers of cellulitis.

We believe, as does the Mayo clinic group, Lockhart-Mummery, and Yeomans that when sloughing, gangrenous, internal hemorrhoids are present, it is usually best to operate immediately. Of course, we grant that this is a moot point and should be judged according to the merits of the individual case.

d. Attempt to make an office procedure of a hospital procedure. The advantages of this method are (1) it is usually easier to get a patient to submit to surgery in the office, and (2) the proctologist more frequently collects for his services, since the patient does not have to pay a hospital bill.

Hirschmann, one of the greatest living proctologists, makes an office procedure of what we regard a major proctological operation. He dispenses with one assistant, four forceps and a weight, giving the exposure of the operative field. The method of exposure in surgery is always important. Proper removal of associated pathological lesions may be missed by Hirschmann's method, due to the distortion of the operative field, certain portions of it being under too great tension. The dangers and difficulties attending a hemorrhage, with a patient away from the office or hospital, are only too apparent. After-care during the first 4 or 5 days is made difficult for the patient and the physician. Hirschmann's method can be mastered by only a few experts. It is not safe in the hands of the majority of proctological surgeons. Hirschmann's easy and sure manner of performing the operation must not unduly sway others to try it; Hirschmann is in a class by himself.

OUR SURGICAL METHOD

Our method can be used by any competent proctologist. We have attempted to overcome such objections as we have noted in other techniques. In addition we wanted to develop a method that would (1) avoid postoperative infection; (2) give absolute control of bleeding, thus insuring no postoperative hemorrhage; and (3) give good final results, with a short, comparatively painless, postoperative course.

Many of the general features of this technique can be applied to other operations about the anus and rectum. However, since hemorrhoidectomy is the most common operation in proctology, this is presented specifically.

Pre-operative care. The usual pre-operative diagnostic work routine is carried out. It consists of digital, anoscopic and sigmoidscopic examinations in all cases, and x-ray and stool examinations as indicated.

The day preceding the operation the patient is kept on a regular diet. About 8:00 p.m., an enema of 1 quart of physiological saline solution is given, with a No. 24 French soft rubber catheter with a velvet eye tip. A hard rubber tip or a colon tube is not used, since this causes trauma at the anal margins. Three grains of sodium amytal are given by mouth at 8:30 p.m.

On the following morning, if the operation is to take place at 8:00 a.m., an enema is given at 6:30 a.m. Six grains of sodium amytal are given by mouth at 6:00 a.m., and 3 grains at 7:00 a.m. One-sixth grain of morphine and 1/150 grain of atropine are given hypodermically at 7:30 a.m. This sedation usually assures a relaxed, unworried patient. Water is forbidden the patient after 5:00 a.m. At 7:30 a.m., the patient voids and then is brought to surgery.

Anesthesia. The anesthesia of choice is caudal and paravertebral block given in the usual manner. Twenty-five to 35 cubic centimeters of one per cent metycaine (formerly novocain) is injected into the sacral canal. Ten cubic centimeters of one half of one per cent metycaine is injected into each of the second paravertebral foramina.

General anesthesia of cyclopropane is used rarely, under two conditions: (1) if the patient refuses caudal-paravertebral anesthesia; (2) in the unusual event that caudal-paravertebral anesthesia fails. This failure is usually due to a misplaced caudal needle. Occasionally it is caused by an attempt to operate upon the patient too

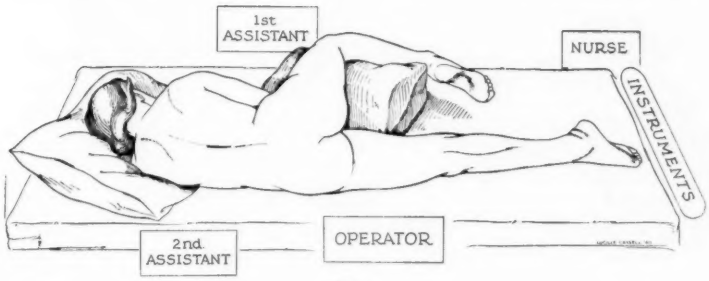

Fig. 1

soon after the anesthetic is given. Sometimes when cyclopropane is given, peri-anal infiltration has been used. One half of one per cent novocain with adrenalin has been the solution injected. Muscular relaxation is aided by the novocain, and the adrenalin tends to prevent some of the excessive oozing which occurs with the cyclopropane. However, the distortion of the tissue caused by the use of a local anesthetic offsets, to our minds, a good deal of the advantages it offers.

The usual rectal operation is emphatically not enough of a major procedure to justify the use of spinal anesthesia. These patients frequently must be up to void within 10 to 12 hours after operation. When a spinal tap has been made, if the patient must arise it will cause disagreeable headaches in a large percentage of the cases.

Position. The position of the patient may be seen in Figure 1. He lies on his left side with the left lower extremity held out in an unbent position, and with a pillow under the flexed right leg. The body falls toward the left side. This exaggerated Sims position gives a good operative exposure without the sacrifice of any convenience of position for the surgeon. It allows the assistants to be of active service, which is difficult when the patient is in a lithotomy position. Overengorgement of the hemorrhoidal area, with the resulting removal of too much tissue is not apt to occur in this posture.

At this time, while the anesthesia is becoming effective, the peri-anal region is shaved. Partial relaxation and anesthesia is present, which makes the procedure less difficult and painful.

Preparation. The perineum and anus are painted with tincture of merthiolate and drapes are placed. The effectiveness of the caudal-paravertebral anesthesia is now tested. A No. 22 needle, 2.5 inches in length, is pushed subcutaneously, the lines of its insertion forming a parallelogram about 1.5 inches from the border of the anus. If the anesthesia is not complete, one half of one per cent solution of metycaine is injected subcutaneously. While the injection is rarely required, the test with the needle is used in all cases to avoid hurting the patient during the operation. For reasons stated we do not like to use local anesthesia. Tincture of merthiolate is then applied to the anus and rectum.

A folded gauze sponge, with a long, attached string, is pushed high up into the rectum, about 3 to 5 inches above the pectinate line (Fig. 2A). This is an important step, as the sponge serves a number of functions: it acts as a block against stool coming down into the operative field; it prevents blood from going upward into the bowel, and by its later return obscuring the operative field or causing the surgeon to search for a source of hemorrhage.

Operation. While the number of hemorrhoids and their position varies, in general there are three main hemorrhoidal masses and one small or minor hemorrhoidal mass. One hemorrhoid is constant in position; it is always located in the right anterior quadrant. The two other masses are located on either side of, and in front of, the posterior commissure, one in the right posterior and one in the left lateral area. The smaller or minor hemorrhoidal mass is usually in the midline, posteriorly. It should always be removed if present, however small it may be. The frequent recurrence of hemorrhoids after an otherwise complete hemorrhoid operation, is usually due to the failure to remove this relatively small mass.

The order of removal of the hemorrhoids does not have to be set, but the following sequence usually is observed (Fig. 2B): first, the left lateral; second, the right anterior; third, the right posterior; and fourth and last, the small hemorrhoidal mass in the midline, posteriorly. The frequent difficulties encountered due to the oozing of blood from the site of a freshly removed hemorrhoid, flowing down and obscuring the field of operation, will be minimized by this sequence.

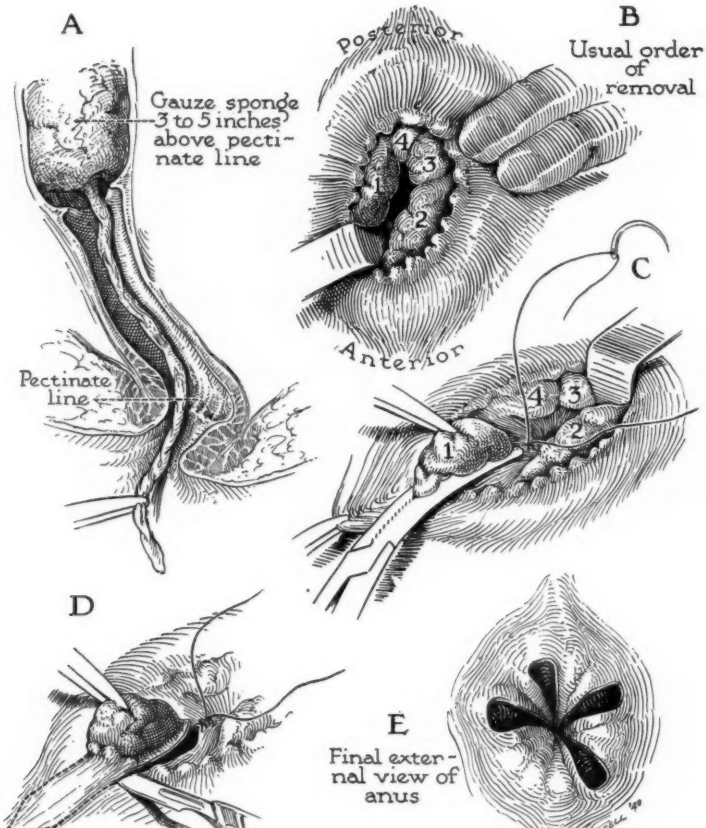

Fig. 2

Exposure of the hemorrhoids is as important as exposure elsewhere in the domain of surgery. This is illustrated in Figure 2C. The skin about 3 inches from the anorectal line is grasped with a 5 inch, curved, Kelly forceps. This forceps is placed in the proper position to make traction on the skin in line with the internal hemorrhoid. The internal hemorrhoid and the external hemorrhoid or skin tag, then present themselves as an anatomically unified defect, and both are removed as one piece of tissue. With this exposure, it is easy to take care of all other pathological tissues near the hemorrhoidal mass. A modified Sims retractor (a flat, rigid, right angled retractor) is placed in the rectum, opposite the hemorrhoid. By pulling on the retractor and the forceps in opposite directions, wide exposure without trauma is obtained. Forcible dilatation of the rectum is never done. Sometimes, with chronic fissures, the anal contracture is sufficient that an anal speculum or the finger is not easily admitted. In these cases, incision is made in the midline posteriorly, cutting down through the sphincter muscle. Painful hematoma and muscle destruction caused by forcible divulsion, frequently resulting in incontinence, are thus avoided.

Lighting is obtained by two means: the first, the standard, overhead, surgical lamp; second a portable surgical lamp placed so as to have the rays directed into the anal canal.

If the hemorrhoids are of the usual small or moderate size, a 5 inch, curved, Kelly forceps is

placed on the hemorrhoid, as illustrated in Figure 2C. The forceps grasp the mucosa, the top reaching toward the upper pole of the hemorrhoidal mass and extending upward ½ to ¾ inch superior to the pectinate line. The lesser curvature of the forceps faces downward. About one-sixteenth of one inch above the tip of the forceps, a quarter length suture ligature of No. 00 chromic catgut threaded on a small, round, full curved needle is passed down through the mucosa just to the muscle, thus picking up and encircling all of the blood vessels. If it is passed too superficially, submucous bleeding occurs, which is very troublesome. If it is passed too deeply, it will include a part of the sphincter muscle, obviously interfering with its function. The suture ligature is tied in the middle. The hemostat is now taken off the hemorrhoid and clamped on the two ends of the suture ligature, which now further acts as a guide for the removal of tissue.

If the hemorrhoid is large, a Tuttle battle-axe forceps is placed slightly above the anorectal line, lifting the upper portion of the redundant tissue in its jaws. The Tuttle forceps serves the same purpose, in a general, limited way, as did the small hemostat. It lifts the hemorrhoidal mass from the musculature of the rectal wall.

The next step is the removal. With a dissecting type blade in a Bard-Parker knife, an incision is made along the lateral surface of the hemorrhoid. The incision starts just below the suture ligature, its shape being seen best in Figure 2D. External hemorrhoids or skin tags, infected crypts, and infected or hypertrophied papillæ are nearly always present. Therefore the incision should extend onto the skin 1.5 to 2 inches below the pectinate line in order to give proper drainage. When the cut is being made, the hemorrhoid is gently held away from the knife by means of a toothed-thumb forceps, held in the left hand of the operator. At the same time the first assistant exerts countertraction in the opposite direction, by means of a similar pair of thumb forceps pulling gently on the rectal mucosa. A second incision is made on the other side of the hemorrhoid, and is of the same order as the first incision except that the ellipse of the curve is reversed. The second incision meets the first one at both extremities to form a parallelogram with two V shaped ends. Incisions on the two sides of the protruding mass should tend to meet underneath it. When the hemorrhoidal tissue is clipped away from the remaining mucosa, there should be a falling together of the edges of the mucosa.

The apex of the "V" on the skin is picked up with the thumb forceps. Dissection is made underneath the hemorrhoid with sharp-pointed scissors, curved, and held on the flat, or with a knife. Dissection extends down to the fascia covering the subcutaneous sphincter and the superficial portion of the external sphincter, but must not ever include the muscles. It must avoid being too superficial, however, or it will fail to include all of the hemorrhoidal vessels. The dissection is continued up to within about ⅛ inch of the transfixion ligature, and then the hemorrhoid is cut off distal to the ligature.

A hemostat is placed across the remaining tissue distal to the ligature, and a second suture ligature is placed beneath this stump. The bleeders now seen are caught with small, curved, Kelly hemostats and ties of No. 00 plain catgut are used. Great care must be taken not to include mucosa or skin in picking up these submucosal bleeders, since this would defeat one of the great advantages of this technique. The transfixion ligatures are the only ones which include mucosa, and their long ends are now cut fairly short. This completes the removal of one hemorrhoidal mass. The other masses are removed in the same manner.

The final steps in the operation include the following: When the bleeding is controlled thoroughly, the sponge with its attached string is pulled out. If additional sponges had to be pushed up above the operative area to block seepage of liquid stool, these are delivered when the main gauze sponge is removed. All blood is kept from finding its way into the upper portions of the rectum and rectosigmoid. If a sponge should be left it will pass with the stool. However, since it usually acts as a block to an easy passage, this should be avoided.

A Hill type speculum is then inserted into the anal canal and the freshly operated upon areas are inspected. All blood is cleaned from the anorectal area in order that a clean view of the field may be obtained and, since all apparent bleeding is controlled, if any postoperative bleeding occurs the operator will know that a fresh hemorrhage has taken place. This point has been stressed by the Mayo clinic group.

With the speculum left inserted, it is made certain that all visible pathological lesions of the rectum have received proper care. A sponge saturated with 2 per cent tincture of merthiolate is placed in the anal canal, and the speculum is withdrawn. The sponge is removed after 1 minute has elapsed. The speculum is replaced and any excess of tincture of merthiolate remaining in the rectum is taken up with a dry sponge.

One ounce of eucupin ointment is injected into the anorectal canal by means of a glass syringe

with a catheter tip. A firm, gauze pressure dressing is laid over the anus and held in place by means of two strips of adhesive. The dressing is pulled tightly against the perineum. Anesthetic ointments do not prevent postoperative pain to any appreciable extent. Gentleness in handling the tissues and adequate care of all visible anorectal lesions have been found to be more effective.

The following precautions may be noted: Tincture of merthiolate is used freely throughout the operation. Each hemorrhoidal area is painted before excision, and the exposed area remaining after removal of the hemorrhoid is swabbed with the antiseptic.

Caution is always used to leave strips of mucosa between the areas of excision. If this is not done, the difficulties encountered with the Whitehead operation are prone to appear.

Prolapsing, thrombotic, internal hemorrhoids are replaced. They are held in position by means of an external, plug, pressure dressing. No operative work is attempted while they and the surrounding tissues are in an edematous and distorted condition.

Thrombotic, external hemorrhoids receive the accepted, usual care. An elliptical incision with a scissors is made. Shelling out of the clot is done, after which care is used to look for any bleeding. No other surgery is done at the time.

The associated, visible, pathological tissues receive thorough care. Infected crypts must be excised. Enlarged, frequently infected papillæ are usually removed, along with the hemorrhoidal masses. These are two of the most commonly found, associated pathological conditions.

Fissures and ulcers have been discovered in about 32 per cent of the patients presenting themselves for hemorrhoidectomy. They are generally situated in the midline, posteriorly. Chronic fissures and ulcers should be excised with care to include the indurated, infected tissue surrounding them. A few of the muscle fibers of the external sphincter should be divided. This midline wound must be kept open so that it will heal in from the bottom. This is aided by placing a strip of vaseline, gauze tape, one-half inch wide, one and one-half inches long, and of one thickness, flat in the wound. This will remain in place for about 3 days and will not interfere with bowel movements. This packing is placed at the final exposure of the field with a small Sims speculum. Occasionally an early fissure is seen. With these, we merely cut down through the fissure and a few of the muscle fibers of the external sphincter.

If any small fistulas are present, these receive care after other operative procedures are completed. If they are large and extend onto the perineum, the operation is performed with the patient in the lithotomy position. While this position is the best for large fistulas and anterior fistulas, nevertheless it is not so good for the excision of hemorrhoids, due to the engorgement of the field.

ADVANTAGES OF THIS TECHNIQUE

1. Postoperative recurrences requiring secondary operation are a rarity. We feel that the removal of the fourth small midline posterior hemorrhoid along with the associated pathological process which is frequently present in this area, contributes greatly to this success. This is the first time this fact has been called to the attention of surgical proctologists.

2. Postoperative infection is rare following the use of this technique. All traumatized tissue is removed. No undermined or pocketed mucosa or skin is left behind. There is no sewing or tying together of edges of undermined mucosa or skin, and no traumatized mucocutaneous teats are sewed or tied to mucosa. The avoidance of oversewing makes more feasible the removal of associated anorectal disease, since ample drainage is present. The large incision onto the skin and the shape of the incision aid the drainage. No irritating rectal plug or larger anal packs are used.

3. Hemorrhages do not occur with this method. The suture ligatures insure unusually good control of bleeding.

4. A short, comparatively painless, postoperative course follows this operation. Modern, postoperative treatment has decreased the pain after almost any type of hemorrhoidectomy. The regimens in general avoid the constipating measures, use a quite liberal diet, avail themselves of the advantages of heat, and have the patient semi-ambulatory at an early date. This method is unusually painless due to the care taken not to tie the skin edges together.

5. Good final results are obtained. A good postoperative result implies that the patient must have good control, that all visible pathological lesions of the anorectal area have been removed, that the anal outlet must be of normal caliber, and if possible that a good cosmetic result has been obtained.

This operation gives these desired good final results for a number of reasons:

1. The sphincter muscle is not damaged. It is not traumatized by forceful dilatation. Stretching causes hemorrhage and infection with resultant scarring and weakening. There is no danger of cutting the sphincter muscle, since the hemor-

rhoid is lifted away from it, thus making separating dissection an easy matter. In the case of the small hemorrhoid this is accomplished by the use of the crushing clamps, and in the case of the large hemorrhoid by the application of the battle-axe forceps.

2. Unusually adequate exposure is afforded by the combination of a good anesthetic and the use of a retractor and a Kelly forceps to make traction in opposite directions.

3. Associated pathological lesions are easily corrected. The operative field is divided into units, and the work is thoroughly completed in a given unit before that in another is started.

4. The fourth hemorrhoid is removed.

5. No circular incisions around the anal canal are made. Such incisions tend to cause strictures.

TREATMENT AFTER THE OPERATION

Upon return from the operating room, the patient is given ½ grain of codeine and 10 grains of aspirin, by mouth. This is repeated every 4 hours, as needed. An order is left for ¼ grain of morphine by hypodermic injection if necessary, but less than 5 per cent of the patients require any morphine after operation. The patient lies on the ventral surface of the body for 1 hour after operation, to cut down venous stasis and bleeding in the operative area. He may then assume any position that he wishes. If necessary, he is allowed to get up to void within 10 to 12 hours following the operation. Catheterization should not be done too soon on these patients. If catheterization is once started it is difficult to stop. Most of these patients will void if given a little time, and the Mayo clinic group advocate the avoidance of catheterization for at least 18 hours after operation. Loosening of the pressure bandage is often valuable in aiding the patient to void during the first 24 hours.

On the first day after operation, that is the day after the operative day, the patient is allowed up and about. On sitting up, the patient had best use a hard chair, as this will be found more comfortable than a cushion which would push up on the operative area. If the patient wishes to sit in a reclining chair, two small pillows may be placed side by side with a four inch space between them, or a rubber ring may be used. A liquid diet is given for lunch and a soft diet for supper the day of the operation, and an all cooked, full bland diet is given starting with the first day after operation.

Difficulty in voiding after the first 24 hours may be relieved by urging the patient to urinate just before leaving the hot sitz bath. Hot sitz baths are given twice daily, and after each defecation, starting on the first day after operation. The utmost precaution should be observed in giving hot sitz baths. An attendant should remain constantly with the patient, because these people, if left alone, frequently get out of the tub quickly and occasionally faint. The sitz baths must be as hot as the patient can bear, and must be continued 15 to 30 minutes to be of any value.

On the first day after operation 1 ounce of petrolagar containing phenolphthalein is given every 4 hours. Starting on the second day after operation 1 ounce of plain petrolagar is given every 4 hours. With these measures the bowel will usually move on the second day after operation and occasionally on the first. Unusually a patient is seen, often of a neurotic type, who will hold back with his stools due to the fear of pain. In these cases an olive oil retention enema and ¼ grain of morphine by hypodermic injection are given. One hour after the oil enema an enema of 1 pint of tap water is given. This olive oil retention enema is not given unless there has been no stool by 4:00 p.m. on the third day after operation. Rarely castor oil will be required. This is undesirable because it produces tenesmus and so completely empties the bowel that there will be no stool again for 48 hours. After the bowel movements have been started, the dose of petrolagar is adjusted to the patient's needs.

It is the personal duty of the surgeon to dress the wound daily. Eucupin ointment on an applicator is applied into the anus and rectum to separate the healing surfaces and to prevent adhesions from forming. The ointment is applied daily after the hot sitz baths, which begin on the first day after operation. Irrigations are painful and have been found unnecessary. If an anal pack was used it is removed in 24 hours unless there was a fissure. A binder is applied. Eucupin provides a non-irritating substance, which helps to keep tissues from adhering to each other. It is not used with the thought of controlling any great amount of pain. If an associated abscess, deep fissure, or fistula was present, beginning on the third day after operation it is painted daily with a cotton tipped applicator which has been dipped into tincture of merthiolate. While this is the most painful part of the entire care it is necessary to help prevent infection in those instances in which the wound is contaminated with pus. Most patients can be dismissed from the hospital on the third or fourth day after operation.

When the patient leaves the hospital, the care can be given by the home physician. The patient

comes to the office each day until the end of the first week after operation, every other day during the second week, twice during the third week, and once during the fourth week. When the patient is having stools comfortably, the well lubricated gloved finger is inserted up the anorectal canal to discover whether any adhesions are present and whether the anus is of sufficient size. If there is a tendency toward contracture, the anus should be gently massaged once every few days until corrected. If this is carried out early contractures do not take place. If a patient is allowed to go without dilatation for several weeks, however, contracture with a small anal aperture may occur which will require a second operative procedure to correct. Following the insertion of the gloved finger or the anorectal dilatation, the rectum and anus are painted with tincture of merthiolate. The sitz baths are continued once daily for 3 weeks. The patient is kept on a cooked bland diet and plain petrolagar for as long a period as seems necessary, this varying from 5 weeks to 3 months.

A final proctoscopic check-up is made by the surgeon 2 or 3 months after operation.

SUMMARY AND CONCLUSIONS

1. A new technique for hemorrhoidectomy is presented in detail. The removal of the fourth small midline posterior hemorrhoid as well as the associated pathology so common in the area is described for the first time in the literature. We feel that removal of the fourth hemorrhoid is one of the most important reasons for the superiority of this technique and for the rarity of secondary operations in our series of cases.

2. The technique described minimizes postoperative infection; it thoroughly controls bleeding; the postoperative period is short and comparatively painless; and most important the final results have been good.

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STRICTURES OF THE MALE URETHRA AND TRICHOMONAS VAGINALIS

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TRICHOMONAS vaginalis is a flagellated protozoan about 8 μ by 15 μ in diameter, which was first described by Donne in 1837. In his original paper he expressed the feeling that gonorrheal infections and Trichomonas were symbiotic. Since 1837 a great deal has been written about this protozoan, many clinicians agreeing to its frequent association with leucorrheal discharges. Its pathogenicity has been commented upon freely. Whether or not Trichomonas are definitely pathogenic is still controversial. Many men are convinced that they do incite purulent inflammatory reactions.

It should be noted that trichomonads have been recovered from many orificial organs of the body, including the vagina, uterus, tubes, Bartholin's and Skene's glands, urethra, bladder, prostate, kidneys, mouth, intestines, and lungs. There is no uniformity of opinion among research and bacteriological workers regarding the morphology, different types of Trichomonas, the number of anterior flagella, and the significance of the undulating membrane. The more frequently mentioned types are the buccalis, hominis, and vaginalis.

As early as 1855 Scanzoni and Koelliker observed trichomonads in over 50 per cent of the women they examined. In 1870 Haussmann reported their presence in 40 per cent of women. De Lee, in 1920, called attention to their frequent occurrence in the vagina and the ease of making the diagnosis with a wet smear. Incidences of 10 to 70 per cent are reported. Allen concludes that Trichomonas vaginitis should be considered as one of the important venereal diseases and feels that sexual intercourse plays an important part in the recurrences. He noted also the frequency of vesical irritability—50 per cent—accompanying Trichomonas vaginitis, and described a specific trigonal lesion. Manwell has discussed the relationship of urinary symptoms in women with Trichomonas vaginitis. Of 33 women so affected, 20 had urinary symptoms. Ten of the 20 had sensitive urethras which bled on instrumentation.

From the Division of Surgery, Department of Urology, Northwestern University Medical School, Chicago, Illinois. Read before the annual meeting, Chicago Urological Society, Chicago, Illinois, May 25, 1939.

Contamination of male urine with trichomonads was first mentioned by Kunstler in 1883. In 1894 Miura found the flagellates in freshly voided urine of a 52-year-old Japanese male whose wife revealed similar protozoa in the vaginal secretions. Marchand, in 1894, found trichomonads in the bladder of a 60-year-old man. Dock also cited a case in the same year. Katsunumma, in 1924, reported finding trichomonads in the urine of a 3-year-old boy. Karnaky, in 1935, reported a case of male Trichomonas urethritis developing after intercourse and persisting for 1 year while in prison.

Additional case reports have been recorded by Capek, Dastidar, Grimm, J. F. Smith of Glasgow, Wright, Rosenthal, Jenkins, Ockuly and many others. Lewis and Carroll, Visher, and Madsen have reported 1 case each of kidney involvement with symptoms due to the Trichomonas. Of these 3, 2 were women, and 1 a young man, age 22. Drummond, in 1936, reported finding by direct smears and cultures trichomonads in 5 husbands whose wives were known to have these protozoa. None of these 5 males had urinary symptoms. Nitschke, also in 1936, stated that in 5, or 12½ per cent, of 40 cases of so called non-specific urethritis, trichomonads were found. He emphasized searching for these flagellates in all such cases in the male. He felt that the difference in incidence of this disorder in males and females was due to the close proximity of the rectum and vagina in women, by which direct contamination could easily occur.

Human trichomoniasis is generally regarded as a local disease, but recent researches by Hees and co-workers indicate that it may be primarily a systemic protean disease. These workers noted that during epidemics there is a parallelism of human and cattle blood infections. Identical Trichomonas strains were found in the blood and intestines of man and cattle. During epidemics, acute cases gave positive blood cultures.

Such an epidemic occurred in Weisbaden between 1934 and 1936. An intermediary species was found, possibly forming the missing link between human and cattle blood species, which is very resistant to outside influences. The fragility of the Trichomonas vaginalis outside the human

body is well recognized and may represent only the terminal phase of its life cycle. Trichomonas genitalis bovis (from uterus of cow) were injected subcutaneously into the thighs of 10 women, 6 of whom developed Trichomonas vaginitis. Oral administration of the bovis type for 5 days resulted in acute trichomoniasis with positive blood and stool cultures and typical Trichomonas vaginalis after 10 days.

Simitch of Yugoslavia established identical human and animal strains in his researches on school children, dogs, and cats. Live Trichomonas hominis have been found in milk and in the excreta of flies.

In recent years, more isolated case reports and small series of Trichomonas cases in males are being recorded in the literature. At this time reports of about 80 cases have been published. The flagellate apparently occurs much less frequently in males than in females. Stuhler at the Mayo Clinic noted that the incidence was fifteen hundred times greater in females. In males, because of the paucity of available clinical material, urological observations have been limited. The reported incidence of Trichomonas prostato-vesiculitis is exceedingly low (Riba, 0.03 per cent; Mayo Clinic, 0.05 per cent).

While trichomonads and gonorrhea have been found and reported simultaneously in females, to our knowledge similar findings in the male have not been previously published. Neither has the apparent relationship between a damaged or strictured urethra and Trichomonas in the male been suggested.

It was of interest to note that in 23 cases of male Trichomonas in our office files, a urethral stricture was present in 14, or 61 per cent. After carefully checking our urethral stricture files, we were surprised to find that Trichomonas had been identified in 10 per cent—14 in 140—of the cases.

The purpose of this paper is to place on record 20 cases of male urethral strictures in whose genito-urinary secretions trichomonads were found. In 2 cases typical intracellular diplococci and trichomonads were repeatedly noted. Summarizing the 20 cases in this study, 14 were private and 6 were from the clinic. Fourteen, or 70 per cent, were married, 4 were single, 1 a widower, and of 1 the status was unknown. Of the 14 who were married, 8, or 57 per cent, of the wives were known to be infected with Trichomonas. Thirteen cases, or 65 per cent, admitted previous neisserian infection, 5, or 25 per cent, denied previous specific urethritis and in 2, or 10 per cent, the information was not obtained. Five, or 25 per cent, gave a history of previous urethral stricture with treatment. The Wassermann tests were negative in all cases. The Kahn was positive in one.

Of the 20 cases, 16, or 80 per cent, presented a mucoid or purulent urethral discharge. Stained smears of the urethral secretions showed nonspecific bacteria in 9, intracellular diplococci in 2, and only pus cells in 5. Trichomonas were found in fresh prostatic secretion in 17, in the urinary sediment in 16, and in the fresh urethral discharge in 11. Pus and shreds in the urine were identified in 18. Pus cells in the seminal fluid were noted in 19: grade 1, 9; grade 2, 6; grade 3, 3; grade 4, 1. On culture 9 cases revealed: streptococci in 3, diphtheroids in 2, Gram positive bacilli in 2, Gram negative bacilli in 2.

The treatment in 18 patients included local and general measures. Oral enteric coated acriflavine tablets of ½ grain were given in 10, sulfanilamide in 3. Local measures included: prostatic massages and instillations in 18; urethral dilatations in 16; preliminary electro-urethrotomy in 3; cystoscopy in 7; intraprostatic mercurochrome injection in 1; weak electrocoagulation of posterior urethra in 1; antiluetic treatment in 1.

Follow-up observations revealed that 15 patients, or 83 per cent, became asymptomatic. In 4, or 22 per cent, however, Trichomonas were still present. Two patients did not return and in 1 symptoms and infection persisted due to a hemipyelonephritis.

SUMMARY

The association and rather high incidence—10 per cent—of male urethral strictures and Trichomonas vaginalis have not been previously described. Two cases are reported in which, in addition to the strictures and trichomonads, intracellular diplococci were found. A damaged or chronically inflamed urethra with lowered resistance seems to favor Trichomonas adaptation and local morbidity and to provide a source for their dissemination.

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EDITORIALS

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REVASCULARIZATION IN SURGERY

THE surgical production of collateral circulation for impaired organs is a subject of current interest which has been extensively studied. Parenchymatous regeneration and organ grafts, which were hailed with so much enthusiasm by surgeons earlier in this century, have failed in practice. To graft organs is very difficult and even if the grafts appear to be successful, the physiological effects are not always constant. Physiological autoregeneration, however, is an established fact. This process is the natural compensatory mechanism of the secretory glands but the regeneration is not always sufficient to maintain adequate function.

Success in transplantation of an organ of which the blood and nerve supplies are cut off is contingent on nutritive juices being delivered by diffusion to the transplanted homologous or heterologous organ. Success perhaps is more likely when tissue of which the blood supply is normally poor is transplanted. Such tissues are bone, stratified epithelium, or undifferentiated tissue such as embryonal mesenchymal layers. The reason for success in transplanting these tissues is partially due to the physical endurance of the cells; and also the potential activity of the tissue.

When the tissue to be used as a graft is highly differentiated, as is the tissue of the glands of internal secretion, nutrition by diffusion is inadequate and the transplant disintegrates and dies. The toxic effect on the graft, exerted by the humors of the host, must be considered principally in relation to heterotransplantation. Homotransplantation offers conditions as nearly ideal as can be found in grafting of tissue. Conditions attending the use of familial grafts are intermediary between the other two.

Diminished or abolished activity of parenchymatous organs often is related to impairment of some kind affecting their nutrition. The lesion may be primary in the peripheral capillary network or secondary to impairment of the parenchymatous cells. In both cases the lesion ends in formation of scar by the growth of granulation tissue, which occludes the blood supply, hinders the venous circulation, disturbs reproduction, and ends in atrophy of parenchymatous cells and senility of the organ.

Function may be suppressed by overgrowth of the connective tissue framework of an organ. The unit of the economy of the organ is a colony of cells living within a mesenchymatous frame; the natural or pathological evolution of the organ is, respectively, senility or disease and either of these kills the indefinite power of reproduction of the host. To

secure integrity of the frame, thus providing for nutrition and reproduction of the involved differentiated cells, constitutes a new problem; experimentation to solve this problem offers a great field for investigators.

Just as it is true that function makes the organ, so also is it true that the organ determines the function; that is, morphology regulates function. In the hive, bees put workers' eggs in the small cells, and drones' eggs in the large cells, for the reason that the bee adapts its function to the shape of the cell. This is illustrative of the function of the cells of differentiated organs. The capillary network of an organ maintains the form of the whole. All the organs are built on the same basic plan and variations in size and arrangement are conditioned by the requirements of function. Every parenchymatous unit is built around a capillary vessel; the vessel is surrounded by mesenchymal embryonal tissue, the morphology of which changes with the function. The architecture of the frame may change but the structural unit always keeps the original characteristics. The frame determines the process of regeneration, as was pointed out by Nageotte in relation to regeneration of bone. The field of bone pathology is very rich in facts supporting this view. Moreover, Beck and Rienhoff's work (1935) on the development of anastomoses between the myocardium and the extracardiac circulation justifies extension of this principle to include the muscular system. Preservation of the original organ-shape has the same importance as the physiological gradient in the development of function.

The great omentum is the largest mesenchymal store in the bodily economy and it plays a very important rôle in compensatory phenomena of adaptation and in regeneration of abdominal viscera. Many highly useful procedures that have been incorporated in routine surgical practice have been inspired by imitation of nature. The so called omental grafts have their physiological background in the collateral circulation that is developed through adhesions between parietal and visceral serous membranes. The widespread subserous venous capillary network relating portal and caval systems, which respectively are visceral and parietal in distribution, develops in every artificial or pathological adhesion between the membrane named. Providential compensatory and protecting mechanisms are endless in the human body; rational surgical procedures must be patterned after them. The extension to other systems, of Beck and Rienhoff's statements relative to the muscular system, offers and promises new horizons in surgery.

Since 1932, in the University of the Republic, at Montevideo, Uruguay, some contributions have been made to the subject under consideration. The round and falciform ligaments of the liver have been separated from the abdominal wall and have been employed as reinforcing tissues in closure of fifty gastroduodenal perforations. Results were better than when omental patches were used. In one instance, eighteen days after the perforation, the opportunity arose to examine histologically the process of healing and the place of the perforation could be detected only in the mucosa. A well developed collateral circulation had brought about complete regeneration of the submucosal layer.

The fibrous, adventitious sac of a hydatid cyst of the lung remains for years as an empty cavity without showing a tendency to healing, because the circulation to the fibrous, pericystic tissue which has undergone condensation is impaired, congested, and blocked; this tissue is very prone to become infected. Pleural adhesion is insufficient to bring about revascularization of the adventitious sac. In

operation for the condition it is the routine, in the university, to draw up the fibrous, adventitious sac through the incision in the thoracic wall, and then to stitch the sac to the parietal thoracic muscles. Drainage of the cavity is maintained and is continued for several days. Histological investigation has proved that muscular capillaries ramify on the fibrous wall of the cavity. This process has a remarkable effect, both in aiding spontaneous closure of the fistula and in furthering healing of the cavity.

Alexander successfully closed bronchial fistulas and cavities which had been the site of suppuration, by inserting a piece of muscle. In surgery of hydatid cysts, opportunities often are offered to judge the value of this practice.

These are the views and contributions of the University of the Republic, in Uruguay to this new surgical conception. Investigation and clinical observation should enlarge the opportunity for its application, especially in the field of endocrinology.

VELARDE PEREZ FONTANA

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

WITHIN the brief space of 15 months Wallace Mason Yater[1] has revised *Fundamentals of Internal Medicine!* The objectives of the original volume—the presentation of the minimum amount of knowledge a medical student or general practitioner should have at all times—are unchanged. The design, section divisions, and contributors of this volume are identical with those of the original edition.

It is obvious that the stimulus for this revision has come from teaching new classes of medical students. It is apparently Yater's purpose that oncoming classes of medical students should have available in a textbook form the latest important knowledge on all medical subjects. The practicality of pursuing this premise is evident. Such a venture would be a year book of internal medicine. Further it may minimize some of the most valuable adjuncts of good medical teaching methods.

Numerous alterations and additions have been made without increasing materially the size of the text. Extensive illustrations have been reduced in size allowing additional text material. Here and there throughout the volume are inserted extensive revisions (serum therapy and chemotherapy of pneumonia). This revision of a distinctly unconventional method of approach in medical pedagogy has much to recommend it. Medical students have and will continue to find it a real help in their initiation into clinical medicine.

FRED FITZ.

THE planographed publication, *Congenital Malformations* by Murphy,[2] analyzes the results of an ambitious statistical study of parental characteristics in families already possessing one malformed offspring. No correlation was established between the occurrence of malformations and the following factors: illegitimacy, social status (except lowest?), illness at time of conception, general unhealth of mother, syphilis, marked age difference of parents, occupation of father, frequency of reproduction, season, placenta previa.

On the other hand, some important positive conclusions are reached. The malformation rate among whites is about twice that in negroes. The chance of occurrence of gross malformation in a family already possessing one defective, is enhanced 25 times. Congenital malformations are increasingly likely with increasing age of the mother; hence later children are more frequently afflicted than earlier ones. It is believed that gross congenital malformations in man arise solely from influences that affect the germ cells prior to fertilization, rather than to environmental influences exerted afterward. In the opinion of the reviewer this latter conclusion is not justified on the basis of the evidence presented. Although germinal inferiority is unquestionably a potent factor, properly timed arrests, due to various environmental causes, is probably also important. If the author is aware of the full argument on this topic, his discussion does not indicate such. Indeed, some of his own data can be brought into line with the theory of arrest. Certain other conclusions have not been subjected to sufficiently severe criticism; for example, that the advanced age of the father is a predisposing factor to malformed offspring rests upon one citation related by a colleague.

To the practitioner the most important advice to be drawn is that the parents of a congenitally malformed child can be warned that their chance of having another malformed child is much greater than with other couples, and that the chance of having another defective child will probably be somewhat further increased if the mother is in her third decade, or more, of life.

L. B. AREY.

THE *Compendium of Regional Diagnosis in Lesions of the Brain and Spinal Cord* by Robert Bing[3] has been in print for the past 30 years and has undergone many changes in its 11 editions. The translation of this book into English will make it extremely popular.

It is written clearly and concisely, and its 275 pages are chock-full of practical points of cerebrospinal localization. There are two parts, that of localization of spinal cord lesions and that of localization of cerebral lesions. In the first part there is a discussion of spinal cord lesions both in the transverse and longitudinal planes. In part 2 lesions in the brain stem, cerebellum, cerebrum, basal ganglia, and hypophysis are discussed. There are 125 excellent illustrations. Many of these are colored. This book is highly recommended to all neurologists and neurological surgeons. It may be of use to medical students as a reference manual.

THEODORE T. STONE.

[1] THE FUNDAMENTALS OF INTERNAL MEDICINE. By Wallace Mason Yater, A.B., M.D., M.S. (in Med.), F.A.C.P. 1st rev. ed. New York, London: D. Appleton-Century Co., 1940.

[2] CONGENITAL MALFORMATIONS; A STUDY OF PARENTAL CHARACTERISTICS WITH SPECIAL REFERENCE TO THE REPRODUCTIVE PROCESS. By Douglas P. Murphy, M.D., F.A.C.S. Philadelphia: University of Pennsylvania Press, 1940.

[3] COMPENDIUM OF REGIONAL DIAGNOSIS IN LESIONS OF THE BRAIN AND SPINAL CORD; A CONCISE INTRODUCTION TO THE PRINCIPLES OF LOCALIZATION OF DISEASES AND INJURIES OF THE NERVOUS SYSTEM. By Robert Bing. Translated and edited by Webb Haymaker. 11th ed. St. Louis: The C. V. Mosby Co., 1940.

THE third edition of Boyd's new standard text, *The Pathology of Internal Diseases*,[1] first published in 1931, with the second edition appearing in 1935, preserves the same organization and format and high excellence as the previous editions.

There are 22 more illustrations, including 4 color plates (353 engravings, 4 colored plates), and 30 fewer pages than in the second edition. The illustrations are of high quality. Almost without exception each fulfills the function of an illustration, namely, to portray the condition under discussion so clearly that it may be comprehended from the picture alone without the necessity of an explanatory legend. The magnifications of all photomicrographs are included, a point particularly appreciated by pathologists, and too often overlooked, even in so-called reference works.

The reduction of pages is accomplished chiefly by the condensation of the 35-page index of the second edition to 17 pages in the present edition. In the opinion of the present reviewer this is an error, for this volume, like its predecessor, will be used by many as a reference book as well as a text to be read systematically. While the present index is good, the other was better.

Text pages are reduced by 11, through the rewriting of certain sections, deletion of older material, and the continued use of small type for subject matter of lesser importance. Adjuvant material such as anatomical considerations and pertinent bacteriological data, adding much to the value of the book, are quite appropriately thus included. The sections most revised are those dealing with the pancreas, blood, kidneys, and vascular system. All subjects are up to date. As previously, the book is organized by organs and organ systems and it is so chaptered.

One finds very little to criticize adversely. The text continues to be written in a clear, engaging style. The clinical aspects and the pathology of disease conditions receive careful consideration. Anatomical data, bacteriology, and physiological considerations are included when these add to the clarification of the disease process.

Diseases are considered from the point of view of living pathology, that is, of deranged physiology, "the dead teach the living." In both the physiology and pathology the text is sound. As in other books by Professor Boyd, illuminating quotations, apt phrases, and historical allusions are liberally and effectively employed to clarify or drive home points, as: rheumatism is a disease "which licks the joints, but bites the heart" (p. 11). Some passages are transformed into exciting literature by these devices, as John Hunter's record of an anginal attack, . . . "I cast my eye on a looking-glass and observed my countenance pale, my lips white, and I had the appearance of a dead man looking at himself" (p. 73).

Selected references are included at the end of each chapter. The usefulness of these could be improved by including the title of each.

This is an excellent book. Highly readable and authoritative, it will be used with profit by general practitioners, surgeons, physicians, and advanced medical students alike. To the clinician it will be of value because of its sound pathology; to the pathologist it will continue as a "must" because of its excellent correlation of the clinical states with morbid anatomy. The medical student beyond his second year and the physician who continues to be a student will also find this book very useful in the correlation of clinical conditions with associated pathology. The advanced student of whatever attainment will find this volume a useful text of both clinical medicine and pathology. To these this book is highly recommended. FRANK B. QUEEN.

BONE and joint tuberculosis is a metastatic focus and is secondary to tuberculosis elsewhere in the body. The importance of resistance against the infection and the need of general hygiene are emphasized in Girdlestone's *Tuberculosis of Bone and Joint*.[2] Aftercare and prophylaxis are important. The etiological factors, particularly the relationship to trauma, as well as the type of tuberculosis, are discussed.

An early definite diagnosis is not always possible, therefore, a provisional diagnosis must be made and treatment carried out as if the diagnosis were definite. Arthrotomy in which a generous section is taken from the joint is a useful diagnostic method, as is removal of a regional gland.

General treatment includes a practical discussion of food. In the discussion on open-air treatment the author emphasizes that benefit is derived from alterations in temperature and action of the winds as well as sun rays. Local treatment means quietness for inflammation, relief of pressure for decalcification and erosion of bone, and relief from friction for softened cartilage.

Children heal better than adults and conservative treatment is carried to its conclusion. In adults, operation is interposed as soon as general and local conditions allow. Operation accelerates healing but does not directly influence lymphatic tuberculosis and is most successful if done when the general condition is at its best and the local lesion is no longer active. The use of intra-articular and extra-articular arthrodesis is discussed. The problem of correction of deformities due to tuberculosis is taken up, pointing out the importance of pre-operative care to decrease the danger of dissemination.

Each articulation is taken up separately. In the discussion of tuberculosis of the hip, the pathology, examination, and a complete differential diagnosis are included before the treatment is discussed. Treatment includes the use of splints and correction

[1] THE PATHOLOGY OF INTERNAL DISEASES. By William Boyd, M.D., LL.D., M.R.C.P. (Ed.), F.R.C.P. (Lond., Dipl., Psych.), F.R.S.C. 3d rev. ed. Philadelphia: Lea & Febiger, 1940.

[2] OXFORD MEDICAL PUBLICATIONS. TUBERCULOSIS OF BONE AND JOINT. By G. R. Girdlestone, M.A., B.M. (Oxon.), F.R.C.S. (Eng.). London: Oxford University Press, 1940.

of deformities. Both conservative treatment and operative treatment are taken up. The operative treatment includes a description of intra-articular and extra-articular operations as well as wide excision, drainage, and amputation. A table of results in non-operative and operative treatment concludes the chapter. The spine, knee, sacro-iliac, ankle, tarsus, shoulder, elbow, and wrist are discussed in a similar manner. Tuberculosis of the metatarsals, metacarpals, and phalanges are also considered. The nerve involvement in case of tuberculosis of the spine (Pott's paraplegia) is explained as to cause, development, prognosis, diagnosis, treatment, and end-results. There is an appendix which describes splints and frames employed by the author. There is also a statistical study of bone and joint tuberculosis from the Shropshire Orthopedic Hospital.

This book is recommended to anyone interested in bone and joint tuberculosis. It represents conclusions based on the author's experience for a period of over 20 years in a hospital which represents the best principles of orthopedic surgery in the care of tuberculosis of the bones and joints. The author is constantly aware that he is dealing with a problem of an invading infection and human resistance. To him, every patient with bone and joint tuberculosis offers an individual problem and he adapts his plan of treatment to suit each case. EMIL D. W. HAUSER.

A SERIES of papers by members of the staff of the Memorial Hospital for Cancer and Allied Diseases in New York have been presented in the volume on *Cancer in Childhood*.[1] The introductory chapter by Dr. James Ewing presents a survey of the cancer problem in childhood. He emphasizes the frequency, the wide variety, and the special characteristics of malignant tumors occurring during the period of childhood. Following this introductory chapter, short papers are devoted to each of the following subjects: malignant tumors of bone; lymphomas, leucemias, and allied disorders; cancer of the genito-urinary organs; gynecological cancer; cancer of the head and neck; tumors of the soft somatic tissues; and blood and lymph vessel tumors.

This collection of papers, written by men of wide experience in their special field, is well worth the attention both of pediatricians and those who are engaged in the study of tumors. STANLEY GIBSON.

WITHIN the limits which the authors have set for themselves the textbook on *Artificial Pneumothorax*[2] is an excellent one. It covers the subject succinctly and clearly. For the beginner in phthisiology it provides an excellent survey and a good point of departure for more intensive reading; and for the veteran it gives a valuable picture of practice and procedure in one of the country's major clinics. For him the later chapters, which present the statistics on results and complications, are particularly important.

It is advisedly that I have stated that the authors have set themselves limits and that I have termed the work a textbook. It is not a monograph or a detailed analysis of the world literature. On the basis of large experience and wide reading they have written a running exposition of current opinion and current conclusions. There are few specific references to the literature, but at the end of each chapter is a practical bibliography.

This method of presentation has many advantages and some shortcomings. It is a welcome relief from the too much documented history. By being brief and didactic it spares the reader the perplexity of conflicting data and contrary conclusions. This easy readability is an asset but one attained at some cost. Uncertainty and controversy are so inextricable a part of science, and current conclusions and present practice are so purely a stage in the evolution toward truth, that it is questionable whether any scientific idea should be presented to a scientist (and medical students and practitioners must be included in this category) without presenting also the data which warrant it and without giving it its aura of uncertainty. Showing present therapy and practice as the product of synthesis and the subject of dispute need not make dull reading,—in fact quite the opposite. It may be less easy, but if selection is judicious, it is infinitely more fascinating, for only when thus shown as offshoots of an organism can ideas be well understood and thoroughly appreciated. JEROME R. HEAD.

THE most recent book[3] of many on the subject of trauma is outstanding on first perusal because of the absence of a single poor illustration. There are 1,040 in all, and every roentgenogram, photograph, fine line sketch, and color plate is beautifully reproduced. The subject matter is based on the management of 47,300 bone and joint injuries cared for on the author's service at the Liverpool Royal Infirmary. Watson-Jones has drawn freely on his own contributions to current surgical periodicals, but has made this book far more than a compend of his own work. Many of the chapters, particularly those on controversial subjects, are handled in an encyclopedic manner, and abundant references to principal contributors in the field are given.

Most of the book is, of course, devoted to the management of regional fractures. The first 150 pages, however, are taken up with general principles. Illustrations and discussion of bone repair, bone necrosis, delayed and non-union, vascular and nerve injuries complicating fractures, adhesions and joint stiffness, open and infected fractures, and the like are found in this chapter. It is here that one finds

[1] CANCER IN CHILDHOOD; AND A DISCUSSION OF CERTAIN BENIGN TUMORS. Edited by Harold W. Dargeon, M.D., F.A.A.P. St. Louis: The C. V. Mosby Co., 1940.

[2] ARTIFICIAL PNEUMOTHORAX; ITS PRACTICAL APPLICATION IN THE TREATMENT OF PULMONARY TUBERCULOSIS. Contributions by Sarnac Lake Physicians to the Studies of the Trudeau Foundation. Edited by Edward N. Packard, M.D., John N. Hayes, M.D., and Sidney F. Blanchet, M.D. Philadelphia: Lea & Febiger, 1940.

[3] FRACTURES; AND OTHER BONE AND JOINT INJURIES. By R. Watson-Jones, B.Sc., M.Ch. (Orth.), F.R.C.S. Baltimore: The Williams & Wilkins Co., 1940.

a fresh and decidedly individual approach to many of these disturbing problems. By being acquainted with the author's viewpoint as expressed in this first chapter, the reader will derive more benefit from the succeeding chapters than if he uses the book as a simple reference on a given subject. This large book covers the field entirely, and with its easy style and abundant illustrations it will be deservedly popular.

JAMES K. STACK.

THE book on *Illustrative Electrocardiography*[1] by Burstein is a small atlas containing approximately 100 plates representing an electrocardiogram or portions of multiple tracings. The first portion of the book illustrates the various waves and normal electrocardiograms followed by a section on the arrhythmias. One section is devoted to block, and another to bundle branch block. There are excellent illustrations of the serial changes in acute coronary thrombosis and also of the effects of digitalis on the electrocardiogram. Fourth lead tracings are shown, illustrating acute coronary thrombosis and also the effects of digitalis.

Most of the electrocardiograms are exceptionally well chosen, and the illustrations are unusually clear and representative. In the latter portion of the book the illustrations for the fourth lead are small and are not up to the standards in the other portions of the text. Some plates show several tracings with arrangement that may be confusing to the student. At the end of the book there is a very good index.

This book will be of interest to beginning students of electrocardiography who wish a small atlas of representative electrocardiograms.

CHAUNCEY C. MAHER.

[1] ILLUSTRATIVE ELECTROCARDIOGRAPHY. By Julius Burstein, A.B., M.D. Originally written by the late Joseph H. Bainton, A.B., M.D., and Julius Burstein, A.B., M.D. 2d ed. New York and London: D. Appleton-Century Co. Inc., 1940.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

LA MANIOBRA ANO PARIETO-ABDOMINAL EN EL ESTUDIO DE LOS PROCESOS AGUDOS DEL ABDOMEN. By Dr. Emilio S. Sammartino. Buenos Aires: "El Ateneo," 1940.

SYNOPSIS OF THE PRINCIPLES OF SURGERY. By Jacob K. Berman, A.B., M.D., F.A.C.S. St. Louis: The C. V. Mosby Co., 1940.

A MANUAL OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY. By Howard Charles Ballenger, M.D., F.A.C.S. Philadelphia: Lea & Febiger, 1940.

THE HEAD AND NECK IN ROENTGEN DIAGNOSIS. By Henry K. Pancoast, M.D., Eugene P. Pendergrass, M.D., and J. Parsons Schaeffer, M.D., Ph.D. Springfield, Ill., and Baltimore: Charles C. Thomas, 1940.

PATHOLOGY. By Eugene C. Piette, M.D. 3d ed. Philadelphia: F. A. Davis Co., 1940.

AN INTRODUCTION TO PHARMACOLOGY AND THERAPEUTICS. By J. A. Gunn, M.A., M.D., D.Sc., F.R.C.P. 6th ed. London: Oxford University Press, 1940.

DISEASES OF THE NERVOUS SYSTEM. By W. Russell Brain, M.A., D.M.(Oxon.), F.R.C.P. (London). 2d ed. London: Oxford University Press, 1940.

THE EMPEROR'S ITCH; THE LEGEND CONCERNING NAPOLEON'S AFFLICTION WITH SCABIES. By Reuben Friedman, M.D. New York: Froben Press, 1940.

FRANK HOWARD LAHEY BIRTHDAY VOLUME. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1940.

MANUAL OF MEDICAL AND SURGICAL EMERGENCIES. Edited by J. C. Geiger, M.D. San Francisco: J. W. Stacey, Inc., 1940.

TRABAJOS DEL DEPARTAMENTO DE MATERNIDAD Y PEDIATRÍA DEL HOSPITAL INTERNACIONAL DE CIUDAD TRUJILLO, REPUBLICA DOMINICANA. Director: Dr. R. R. Cohen. Ciudad Trujillo: Editora Montalvo, 1940.

THE INJURED BACK AND ITS TREATMENT. Edited by John D. Ellis, M.D. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1940.

HISTOPATHOLOGY OF THE PERIPHERAL AND CENTRAL NERVOUS SYSTEMS. By George B. Hassin, M.D. 2d rev. enl. ed. New York and London: Paul B. Hoeber, Inc., 1940.

GYNECOLOGICAL AND OBSTETRICAL PATHOLOGY; WITH CLINICAL AND ENDOCRINE RELATIONS. By Emil Novak, A.B., M.D., D.Sc. (Hon. Dublin), F.A.C.S. Philadelphia and London: W. B. Saunders Co., 1940.

ATLAS OF CARDIOROENTGENOLOGY. By Hugo Roesler, M.D., F.A.C.P. Springfield, Ill., and Baltimore, Md.: Charles C. Thomas, 1940.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

GEORGE P. MULLER, Philadelphia, *President*
EVARTS A. GRAHAM, St. Louis, *President-Elect*

Committee on Arrangements
JOHN A. WOLFER, *Chairman;* CHARLES B. PUESTOW, *Secretary*

PROGRAM FOR THE CHICAGO CLINICAL CONGRESS

THE 1940 Clinical Congress of the American College of Surgeons will be held in Chicago, October 21–25. This will be the thirtieth assembly of this great organization of surgeons of the United States, Canada, and Latin America, the first of these meetings having been held in Chicago in 1910. The surgeons of this city are privileged to be hosts to this meeting for the eighth time since the Clinical Congress of Surgeons of North America initiated this vast educational program which places the primary emphasis upon well organized clinics conducted in the hospitals of a great medical center.

CLINICAL PROGRAM

This year under the leadership of a strong and representative committee, it is planned to present a program of operative clinics and demonstrations in the five medical schools and forty or more approved hospitals which will constitute a highly organized postgraduate experience for all the surgeons who will attend. The operative clinics, demonstrations and exhibits in the hospitals will cover all phases of the clinical activities in the surgical and other departments of these institutions. Representing as it does the interests of all surgical specialists and general surgeons, this Congress will provide a complete and varied program during the five days of the meeting. There will be presentations of the latest advances in diagnostic methods, surgical techniques and operative procedures. Many of these presentations will deal with pertinent subjects related to pre- and postoperative care of the surgical patient.

The major feature of the Clinical Congress, as arranged by the local committee, will be an extensive schedule of operative clinics in which the technique of a wide variety of surgical procedures will be demonstrated in the operating rooms. However, the program of each operative clinic has been planned with the idea of presenting related problems in diagnosis and other special aspects of the management of the surgical condition under consideration. In many of the clinics there will be an opportunity to observe the end-results which have been obtained following specific surgical treatment.

Non-operative clinics and demonstrations are being planned which present the various features of the clinical work which is being done in many of the hospitals and medical schools. Again, all of the special fields of surgery will be represented in this phase of the clinical program, which will provide an opportunity for surgeons engaged in various types of practice to study the latest advances in clinical surgery, as well as important research activities of the various hospitals and medical schools.

Several of the larger hospitals are planning special scientific exhibits. Thus the visiting surgeon will be able to study at first hand much of the clinical and experimental work which is being carried on by the surgeons of Chicago.

The program of each institution is being arranged to cover subjects in general surgery, fractures and other traumas, neurosurgery, orthopedic surgery, thoracic surgery, urology, obstetrics and gynecology, ophthalmology, and otorhinolaryngology. The clinical program, organized under this classification, is being so correlated that those attending the Congress will have ample opportunity to devote their time continuously to clinics dealing with subjects related to the specialty in which they are most interested. Clinics and demonstrations will be held on the afternoon of Monday, October 21, and the mornings and afternoons of each of the four succeeding days.

In order to aid the visiting surgeons in selecting

CLINICAL CONGRESS PROGRAM IN BRIEF

(All sessions at the Stevens Hotel except as noted)

Monday, October 21

10:00 Hospital Conference.
1:30 Panel Discussions (5).
2:00 Clinics in Chicago hospitals.
2:00 Hospital Conference.
2:00 Surgical Film Exhibition, General Surgery.
3:00 Assembly of Initiates, followed by reception—Headquarters of College.
3:30 Panel Discussions (5).
8:00 Presidential Meeting and Convocation.

Tuesday, October 22

9:00 Clinics in Chicago hospitals.
9:30 Hospital Conference.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
10:00 Surgical Film Exhibition, General Surgery.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2).
1:30 Panel Discussions (5).
2:00 Clinics in Chicago hospitals.
2:00 Hospital Conference.
2:00 Symposium on Fractures and Other Traumas.
2:00 Surgical Film Exhibition, General Surgery.
3:30 Panel Discussions (5).
7:00 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
7:30 Hospital Conference.
8:00 Scientific Session, General Surgery.
8:00 Scientific Session, Ophthalmology.
8:00 Scientific Session, Otorhinolaryngology.

Wednesday, October 23

9:00 Clinics in Chicago hospitals.
9:30 Hospital Conference.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
9:30 State and Provincial Judiciary Committees.
10:00 State and Provincial Credentials Committees.
11:00 State and Provincial Executive Committees.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2).
12:00 Meeting of Board of Governors.
1:30 Panel Discussions (5).
2:00 Clinics in Chicago hospitals.
2:00 Symposium on Cancer.
2:00 Surgical Film Exhibition, General Surgery.
2:30 Hospital Conference.
3:30 Panel Discussions (5).
7:00 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
7:30 Hospital Conference.
8:00 Scientific Session, General Surgery.
8:00 Joint Session, Ophthalmology and Otolaryngology.

Thursday, October 24

9:00 Clinics in Chicago hospitals.
9:30 Hospital Conference.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
10:00 Surgical Film Exhibition, General Surgery.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2).
1:30 Annual Meeting, Fellows of the College.
2:00 Clinics in Chicago hospitals.
2:30 Hospital Conference.
3:00 Conference on Graduate Training in Surgery.
3:30 Panel Discussions (5).
3:30 Surgical Film Exhibition, General Surgery.
3:30 National and Regional Fracture Committees.
7:00 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
8:00 Scientific Session, General Surgery.
8:00 Scientific Session, Ophthalmology.
8:00 Scientific Session, Otorhinolaryngology.

Friday, October 25

9:00 Clinics in Chicago hospitals.
9:30 Surgical Film Exhibition, Eye, Ear, Nose and Throat Surgery.
10:00 Surgical Film Exhibition, General Surgery.
2:00 Clinics in Chicago hospitals.
2:00 Group Clinical Conferences (7).
2:00 Surgical Film Exhibition, General Surgery.

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the clinics which they desire to attend, the *Daily Clinical Bulletin* will present the program according to the foregoing classification. The complete details of the clinical program for the following day will be posted each afternoon in the form of bulletins at headquarters in the Stevens Hotel. The *Daily Bulletin* will be distributed in printed form each morning during the Congress.

EVENING SCIENTIFIC SESSIONS

The opening scientific session of the Clinical Congress will be held on Monday evening in the Ballroom of the Stevens Hotel, when the Presidential Meeting and Convocation will be combined. The new officers of the College will be inaugurated and the 1940 class of initiates received into fellowship. Distinguished surgeons from foreign countries will then be introduced, following which Dr. George P. Muller, of Philadelphia, will deliver the presidential address. "Principles of Colonic Surgery" will be the subject of the sixth annual oration on surgery to be delivered by Dr. Fred W. Rankin, Lexington, Kentucky.

Scientific meetings will be held on Tuesday, Wednesday, and Thursday evenings at the headquarters hotel, when distinguished members of the profession of international prominence will address the assembled guests of the Congress. The speakers and subjects of these addresses have been carefully selected in order to assure presentations which will be of interest to those who are practicing in all of the special fields of surgery. This program has been prepared by the Board of Regents of the College in order to give consideration to as many of the latest advances in general surgery and the surgical specialties as possible.

These scientific papers will be of such importance as to represent outstanding contributions to the surgical literature.

PANEL DISCUSSIONS

Panel discussions have met with such decided success at the Clinical Congress and in the sectional meetings of the College that the schedule this year will include an elaboration of this phase of the program as it is arranged for Monday, Tuesday, Wednesday, and Thursday afternoons. Recognized authorities in special fields are cooperating with the College as leaders and collaborators of these panels; and the topics which have been selected cover many pertinent subjects. This type of program permits more informal discussion of the subject than would be possible in the larger meetings. The plan provides that the leader will present his subject within a ten minute period; collaborators will then discuss various phases briefly, after which general discussion from the floor will be encouraged.

GROUP CLINICAL CONFERENCES

A new feature of considerable interest to all surgeons attending the Congress will be the series of group clinical conferences which is being arranged for Friday afternoon. Nine special fields of surgery have been selected and an important subject of current interest will be briefly presented in each conference. These special fields are: Fractures and other traumas, neurosurgery, obstetrics and gynecology, orthopedic surgery, plastic surgery, thoracic surgery, and urology. The leaders are selecting the subjects which will be presented. They will direct discussions from the floor and visitors attending the meeting will be invited to present any problem with which they may be concerned—one that is related to the special field under discussion. Recognized authorities will endeavor to answer these questions and offer as much helpful advice as possible. Everyone should benefit by participating in a consultation conference of this nature.

SYMPOSIUM ON CANCER

The Cancer Committee of the College has done outstanding work in furthering the development of cancer clinics in hospitals and providing for the registration of cured cases of malignant disease in the cancer archives. The chairman of this committee will give a brief review of these activities of the College in opening the symposium on cancer which will be held Wednesday afternoon. Other subjects of practical interest which will be presented include various problems in the diagnosis and treatment of "Neurogenic Sarcoma" and "Pelvic Cancer." In addition, one speaker will discuss "The Present Status of Carcinogens and Hormones in Cancer Research" and another will lead a discussion on the subject of "Radiation Damage of Tissue and Its Repair." In this symposium the surgeon, the pathologist and the radiologist will discuss important aspects of the cancer problem which are of current interest.

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

In this symposium, to be held on Tuesday afternoon, the Chairman of the committee will make a brief statement concerning the work which has been done during the past year by the national and regional fracture committees. The scientific papers to be presented at this meeting will emphasize practical subjects dealing with different types of fractures which present special problems in management and treatment. One of the papers will deal particularly with "Undergraduate Education in Fractures and Other Traumas." The program will be of interest to all surgeons who engage in this type of practice and will afford an opportunity for them to learn different viewpoints regarding the handling of many clinical problems.

OPHTHALMOLOGY AND OTOLARYNGOLOGY

A new departure from the proceedings of the Congress in former years is being arranged in ophthalmology and otolaryngology. Separate sessions scheduled for Tuesday and Thursday evenings will include panel discussions and symposia on important subjects in these fields. The leaders and a group of outstanding surgeons will direct the discussions in these meetings so as to cover many phases of a general subject in each of the specialties and a number of different viewpoints will be expressed. On Wednesday evening, there will be a joint session of ophthalmologists and otolaryngologists with a symposium on the important subject "Primary Treatment of Injuries about the Face." An ophthalmologist, an otolaryngologist, a maxillofacial surgeon, and a neurosurgeon will cover all aspects of this subject. This type of program promises to attract wider interest than the usual presentation of formal papers.

Clinics in Chicago hospitals each morning and afternoon for the visiting ophthalmologists and otolaryngologists will demonstrate surgical work of a wide variety. On Tuesday, Wednesday, and Thursday mornings, in addition to the hospital program, there will be clinical conferences for each group of specialists at the headquarters hotel. These are being arranged so that the leader will

briefly survey the field for discussion in a ten minute period. The large meeting of surgeons will then be broken up into small groups, limited to 20 and under separate leaders, where there will be an opportunity for everyone to ask questions and participate in the discussions. Each visitor will select in advance the small section of the meeting which he wishes to attend. In this manner, the general subject may be discussed thoroughly and each will benefit by the experience and different viewpoints of his colleagues. The success of these conferences is assured. The morning and evening meetings will be preceded by the showing of selected surgical motion picture films.

GRADUATE TRAINING IN SURGERY

Following the annual meeting of the fellows, to be held on Thursday afternoon, there will be a panel discussion on graduate training in surgery. The chairman of the committee of the College, which represents the surgical specialist as well as the general surgeon, will summarize the work which has been done during the past three years. Following this report, there will be one formal paper dealing with plans of graduate training in surgery in hospitals which have no direct medical school affiliations. The teaching methods employed in educational programs in institutions of this type will be described in detail. The meeting will then be turned over to informal discussions from the floor. The leader will direct these discussions on the problems presented in developing graduate training in hospitals where undergraduate teaching may be limited or entirely lacking. This program should elicit the interest of all hospital executives, surgeons, and educators who are concerned with the future standards of surgery in the United States and Canada.

ASSEMBLY OF INITIATES

On Monday afternoon in the Auditorium of the American College of Surgeons headquarters at 40 East Erie Street, there will be held the Assembly of Initiates. This meeting will be presided over by Dr. George P. Muller, President of the College, and Dr. Arthur W. Allen, Boston, Vice-Chairman of the Board of Regents. Dr. Bowman C. Crowell and Dr. Malcolm T. MacEachern will discuss "The Program of the American College of Surgeons." Initiates will then recite the Fellowship Pledge and greetings will be extended by Dr. Evarts A. Graham, St. Louis, President-Elect of the College. Closing remarks will be made by Dr. Irvin Abel, Chairman of the Board of Regents. Following the adjournment of this assembly, a reception for the initiates, their wives and friends will be held by the officers and Regents of the College.

MEETING OF NATIONAL AND REGIONAL FRACTURE COMMITTEES

The meeting of the National and Regional Fracture Committees will be held on Thursday afternoon, when this large body of surgeons will assemble for a discussion of the activities of the respective groups. Working in co-operation with the American Red Cross, other local organizations and public officials, these committees have exerted great influence in providing improved methods and facilities for the transportation of the injured. There has also been a concerted effort to improve the treatment of fractures in the hospitals of all communities.

ANNUAL MEETINGS OF COMMITTEES

The annual meetings of the State and Provincial Judiciary, Credentials, and Executive Committees will be held on Wednesday morning. These committees have an important function to perform in the activities of the College. The Credentials Committees and the Committees on Applicants provide an organization which constitutes one of the largest and most carefully deliberate accrediting bodies which exist in the medical profession. Through this organization, the standards of fellowship are maintained and each fellow of the College has a definite responsibility in this work. All members of each of these committees are urged to attend these important sessions.

ANNUAL MEETING OF THE FELLOWS

The annual meeting of the fellows will be held on Thursday afternoon. The American College of Surgeons has been a potent force which has not only materially raised the professional and ethical standards of surgery, but has also promoted good hospitalization and general improvement in the practice of medicine in the United States and Canada. These activities have received wide recognition by professional groups and the public as well. Each individual fellow of the College has a part in this work and may extend its influence materially in his local community. Hospital standardization alone offers him unlimited opportunity to provide better medical care for his patients in the hospital in which he works through continuous progress in applying the principles of the minimum standard. Approval of hospitals, plans for graduate training in surgery, cancer clinics, medical service in industry, medical motion pictures, the Clinical Congress, and sectional meetings present a vast educational program.

The annual meeting of the Fellows affords the officials of the College an opportunity to report on the activities of the organization and to receive suggestions from those who have made possible the conduct of these activities. Every fellow of the American College of Surgeons will want to attend this important meeting and participate in the program.

MEDICAL MOTION PICTURES

An enlarged program of surgical motion pictures will be presented at headquarters which will include the latest available films on a wide variety of subjects of interest to the surgeon. The schedule will be arranged so as not to conflict with either the clinical program at the hospitals or the scientific sessions and will include both sound and silent, standard and color films which have been approved by the College Committee on Medical Motion Pictures.

HOSPITAL CONFERENCES

The twenty-third annual hospital standardization conference will open the Clinical Congress with a meeting at the headquarters hotel on Monday morning at 10:00 o'clock. The report of the 1940 hospital standardization survey—official announcement of the list of approved hospitals and hospitals approved for graduate training in surgery—will be made at this session. In addition, there will be a very interesting discussion on "Medical Preparedness in the Case of a War Emergency." The Surgeon General of the United States Navy, Admiral Ross T. McIntire, will give a brief address and E. W. Jones, Superintendent of the Albany Hospital, Albany, New York, will present the plan which has been developed in this hospital for organizing the entire personnel in case of such an emergency.

The afternoon session on Monday will be given over to significant discussions on convalescent care. The subject will be presented from the sociological and economic viewpoints by leading surgeons and others who have had experience with this problem in their respective communities. Fundamental principles in organizing for adequate convalescent care of the surgical patient will be emphasized.

The Tuesday morning session of the hospital conference will include three important panel discussions: "The Control of Surgery," led by Dr. Harold Foss of Danville, Pennsylvania; "The Control of Infections," led by Dr. Frank L. Meleney of New York; and "The Physical and Administrative Hazards of Anesthesia," led by Dr. John S. Lundy of Rochester, Minnesota. These important panels will be interesting to both hospital visitors and fellows of the College. The afternoon session on Tuesday will be devoted to a discussion of "Nursing in the Hospital." There will be a discussion of "The Essentials of Good Nursing Service," followed by a panel discussion on "Administrative Costs of Nursing in Hospitals." A keenly debated subject will also be brought up for discussion, namely, "The Accrediting of Schools of Nursing Education." There will be a panel round table conference on Tuesday evening which will cover all phases of organization, professional practice and public relations. This session should arouse the interest of all hospital visitors, particularly those who are charged with the responsibility of administration in the small hospital.

On Wednesday morning, at a joint session with the American Association of Medical Record Librarians, there will be a discussion of "The Adoption of the Standard Nomenclature," "The Proposal of a Surgical Nomenclature," "The Punch Card System," and "The Relationship of the Medical Record Librarian to the Medical Staff." These subjects present pertinent problems in the organization of a medical records department in the hospital. The afternoon session on Wednesday will be devoted to demonstrations and group conferences covering administrative practices and professional procedures held in ten of the leading approved hospitals of the Chicago area. On Wednesday night the panel discussions will provide a means for presenting specific questions on hospital procedures to a group of leaders in the hospital field.

On Thursday morning the panel discussion will deal principally with the care of tuberculosis patients in general hospitals, the relation of the administration of the hospital to organized medicine, medical staff organization, health service for hospital personnel, and the cost of hospital care. The conference will close on Thursday afternoon with a series of group conferences and demonstrations dealing with administrative and professional practices in leading Chicago institutions.

A new feature has been added to the hospital conferences this year—on Tuesday, Wednesday, and Thursday mornings there will be held breakfast conferences commencing at 7:45 a.m. and ending promptly at 9:15. While breakfast is being served, there will be an opportunity for those attending to participate in a free and animated discussion of each subject under consideration. Each morning ten minutes will be allowed for the speaker to present his subject, following which each one in attendance will be called upon to give

his reaction to the matter under discussion. The subjects will be: Tuesday morning "The Hospital Administrator's Program for Self Development"; Wednesday morning "The Medical Records Librarian's Program for Self Development"; Thursday morning "The Public Relations Program of Your Hospital." As the attendance at these meetings will be limited, there will be arrangements for advance registration.

Perhaps the most important feature of the hospital conference will be the daily consultation service which will be provided. The information desks will be in charge of personnel of the Hospital Standardization Department of the College who will arrange for consultations on hospital problems with the field representatives of the organization and others who will be able to give authoritative information on various subjects. All who attend the hospital conferences are urged to present their particular problem and avail themselves of this service.

ADVANCE REGISTRATION

The hospitals and medical schools of the Chicago area afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, attendance at the Congress will be limited to the number that can be comfortably accommodated at the clinics. The limit of attendance will be based on a survey determining the available facilities in the participating institutions. It is expected, therefore, that surgeons who wish to attend the Congress will register in advance.

A registration fee will be required in order to provide funds with which to meet expenses of the meeting. A formal receipt will be issued to each surgeon registering in advance which will be exchanged for a general admission card upon presentation at headquarters during the Congress. This card, which is not transferable, must accompany all requests for clinic tickets and be presented for admission to the scientific sessions.

A resolution adopted by the Board of Regents provides that the registration fee for fellows of the College and endorsed junior candidates shall be $5.00; that no fee for the 1940 Clinical Congress shall be required of initiates (class of 1940); that the fee for surgeons who are not Fellows, attending as invited guests of the College, shall be $10.00.

As in previous years, admission to clinics and demonstrations in the hospitals and certain of the scientific meetings at headquarters will be controlled by means of tickets. This plan provides for the distribution of visiting surgeons at the various clinics and other meetings and helps to insure against overcrowding. The number of tickets issued for any clinic will be limited to the capacity of the room in which the clinic is held. Visiting surgeons are urged to co-operate in making the clinic ticket plan a success.

HEADQUARTERS—TECHNICAL EXHIBITION

Headquarters for the Congress will be established at the Stevens Hotel where there are unusual facilities for accommodating the Congress. All of the public rooms have been reserved for conferences, registration, ticket bureaus, clinic bulletins, executive offices and scientific exhibits. Thus, all activities of the Congress, except the clinical program, will be located under one roof.

The technical exhibition together with the registration desk will be located on the lower floor of the Stevens Hotel in the large exhibition hall. Leading manufacturers of surgical instruments and supplies, sutures, dressings, pharmaceuticals, operating room equipment, x-ray apparatus and hospital equipment of all kinds, as well as publishers of medical books, will be represented in the exhibition. It will provide for the visiting surgeons an opportunity of carefully inspecting the finest modern products of all those industries which are aiding the work of the surgeon and the hospital.

CHICAGO HOTELS AND THEIR RATES

In addition to the headquarters hotel, the Stevens, there are several first-class hotels within short walking distance of headquarters, providing ample hotel facilities at reasonable rates. It is suggested that reservation of hotel accommodations be made at an early date. The following hotels are recommended by the Committee:

	Minimum Rate with Bath	
	Single	Double
Auditorium, 430 S. Michigan Ave.	$2.50	$4.00
Bismarck, 171 W. Randolph St.	3.50	5.00
Blackstone, Michigan Ave. at 7th St.	4.00	6.00
Congress, 500 S. Michigan Ave.	3.00	5.00
Drake, Michigan and Lake Shore Drive	4.00	6.00
Harrison, 57 E. Harrison St.	2.00	3.00
Lake Shore Drive, 181 Lake Shore Drive	4.00	6.00
LaSalle, 10 N. LaSalle St.	2.50	4.00
Morrison, 79 W. Madison St.	2.50	4.00
Palmer House, 15 E. Monroe St.	3.50	5.00
Sherman, 106 W. Randolph St.	2.50	4.00
Stevens, 720 S. Michigan Ave.	3.00	4.50

COMMITTEE ON ARRANGEMENTS

EXECUTIVE COMMITTEE

John A. Wolfer, Chairman
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Fred L. Adair
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HOSPITALS AND REPRESENTATIVES

Alexian Brothers—George L. Apfelbach.
Augustana—Oscar E. Nadeau
Albert Merritt Billings and Bobs Roberts Memorial—Dallas B. Phemister.
Chicago Lying-in—Fred L. Adair.
Chicago Memorial—Peter S. Clark.
Children's Memorial—Albert H. Montgomery.
Columbus—Daniel A. Orth.
Cook County—Marshall Davison.
Evangelical—G. Henry Mundt.
Evanston—Frederick Christopher.
Grant—A. G. Zimmerman.
Henrotin—John A. Graham.
Holy Cross—John F. Ruzic.
Illinois Eye and Ear Infirmary—Thomas D. Allen.
Illinois Masonic—Walter C. Bornemeier.
Jackson Park—Arrie Bamberger.
Loretto—James A. Valentine.
Mercy—Charles F. Sawyer.
Michael Reese—Ralph B. Bettman.
Mother Cabrini—Eugene J. Chesrow.
Mount Sinai—Jacob M. Mora.
Municipal Tuberculosis Sanitarium—Richard Davison.
Norwegian-American—Harold A. Sofield.
Passavant Memorial—Loyal Davis.
Presbyterian—Vernon C. David.
Ravenswood—Darwin B. Pond.
Research and Educational—Warren H. Cole.
St. Anne's—George F. Thompson.
St. Anthony de Padua—Frederick W. Slobe.
St. Bernard's—John G. Frost.
St. Elizabeth's—Martin G. Luken.
St. Joseph—Hugh McKenna.
St. Luke's—Selim W. McArthur.
St. Mary of Nazareth—George M. Mueller.
Shriners' for Crippled Children—Beveridge H. Moore.
Swedish Covenant—Karl L. Vehe.
Veterans Administration—Paul F. Brown.
Washington Boulevard—Arthur R. Metz.
Wesley Memorial—Raymond W. McNealy.
Women and Children's—Maude H. Winnett.

PROGRAMS FOR EVENING SESSIONS

Presidential Meeting and Convocation—Monday, 8:00 p.m.—Ballroom, Stevens Hotel

GEORGE P. MULLER, M.D., Philadelphia; President, American College of Surgeons, Presiding.
Processional—Officers, Regents, and Honorary Guests.
Invocation.
Address of Welcome. JOHN A. WOLFER, M.D., Chicago; Chairman, Committee on Arrangements.
Introduction of Foreign Guests.
Address of Retiring President. GEORGE P. MULLER, M.D., Philadelphia.
Inauguration of Officers:
President: EVARTS A. GRAHAM, M.D., St. Louis.
First Vice President: OLIVER S. WAUGH, M.D., Winnipeg.
Second Vice President: ALBERT O. SINGLETON, M.D., Galveston.
Presentation of Initiates for Fellowship. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents.
Conferring of Fellowships by the President. EVARTS A. GRAHAM, M.D., St. Louis.
Conferring of Honorary Fellowships. The President.
Medical Records Prize Award.
Annual Oration on Surgery: Principles of Colonic Surgery. FRED W. RANKIN, M.D., Lexington.

Tuesday, 8:00 p.m.—Ballroom, Stevens Hotel

EVARTS A. GRAHAM, M.D., St. Louis; President, American College of Surgeons, Presiding.
Surgical Complications in Pregnancy. JOHN R. FRASER, M.D., Montreal.
Aseptic Resections in the Gastrointestinal Tract, with Special Reference to Resection of the Stomach. OWEN H. WANGENSTEEN, M.D., Minneapolis.
Symposium: The Esophagus.
Observations on the Diagnosis and Treatment of Caustic Burns, Cicatricial Stenosis and Achalasia. GABRIEL TUCKER, M.D., Philadelphia.
Surgical Treatment of Achalasia. ALTON OCHSNER, M.D., and MICHAEL E. DEBAKEY, M.D., New Orleans.
Recent Progress in the Surgical Treatment of Carcinoma of the Esophagus. WILLIAM E. ADAMS, M.D., Chicago.

Wednesday, 8:00 p.m.—Ballroom, Stevens Hotel

OLIVER S. WAUGH, M.D., Winnipeg; Vice President, American College of Surgeons, Presiding.
Sulfonamide Therapy as an Aid to Surgery. JOHN S. LOCKWOOD, M.D., Philadelphia.
Oration on Traumatic Surgery: Treatment of Traumas of Skin and Subcutaneous Tissues. FREDERIC W. BANCROFT, M.D., New York.
The Civilian Surgeon in War. BRIGADIER GENERAL RAYMOND F. METCALFE, M.C., U.S.A., Washington.
A Brief Statement of the Work of the National Research Council Committee on Surgery in Connection with the Program of Military Preparedness. EVARTS A. GRAHAM, M.D., St. Louis.

Thursday, 8:00 p.m.—Ballroom, Stevens Hotel

ALBERT O. SINGLETON, M.D., Galveston; Vice President, American College of Surgeons, Presiding.
Gastrojejunocolic Fistula. DAMON B. PFEIFFER, M.D., Philadelphia.
Some Factors Influencing the Curability of Cancer of the Stomach. THOMAS F. MULLEN, M.D., San Francisco.
The Management of the Neurogenic Bladder in Traumatic Lesions of the Spinal Cord and Cauda Equina. REED M. NESBIT, M.D., Ann Arbor, Mich.
Heparin in Thrombosis and Blood Vessel Surgery. D. W. GORDON MURRAY, M.D., Toronto.

PROGRAMS FOR EVENING SESSIONS

OPHTHALMOLOGY

Tuesday, 8:00 p.m.—Stevens Hotel

Panel Discussion: Surgery of Squint. WILLIAM THORNWALL DAVIS, M.D., Washington, Presiding.
Collaborators: F. BRUCE FRALICK, M.D., Ann Arbor, Mich.; SANFORD R. GIFFORD, M.D., Chicago.

Thursday, 8:00 p.m.—Stevens Hotel

SANFORD R. GIFFORD, M.D., Chicago; Chairman, Advisory Council for Ophthalmology, Presiding.
Symposium: Advances of the Past Ten Years in Ophthalmic Surgery.
Principles of Cataract Surgery. JOHN H. DUNNINGTON, M.D., New York.
Glaucoma Surgery. ALGERNON B. REESE, M.D., New York.
Retinal Detachment Surgery. DERRICK T. VAIL, JR., M.D., Cincinnati.

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Wednesday, 8:00 p.m.—Stevens Hotel

HARRY S. GRADLE, M.D., Chicago; Regent, American College of Surgeons, Presiding.
Symposium: Treatment of Injuries about the Face.
The Treatment of Injuries of the Globe and the Lids, and the Orbit. EDMUND B. SPAETH, M.D., Philadelphia.
Primary Care of Injuries of the Face and Jaws. V. H. KAZANJIAN, M.D., Boston.
The Late Care of Severe Injuries of the Face and Jaws. EARL C. PADGETT, M.D., Kansas City, Mo.
Neurosurgical Aspects of Facial Injury. HARRY H. KERR, M.D., Washington.

OTORHINOLARYNGOLOGY

Tuesday, 8:00 p.m.—Stevens Hotel

SAMUEL SALINGER, M.D., Chicago; Member, Executive Committee on Arrangements, Presiding.
Symposium: Meniere's Disease.
Medical Treatment of Meniere's Disease. HAROLD G. TOBEY, M.D., Boston.
The Use of Histamine in Meniere's Disease. BAYARD T. HORTON, M.D., Rochester, Minn.
The Surgical Treatment of Meniere's Disease. WALTER E. DANDY, M.D., Baltimore.
General Discussion.

Thursday, 8:00 p.m.—Stevens Hotel

PERRY G. GOLDSMITH, M.D., Toronto; Chairman, Advisory Council for Otorhinolaryngology, Presiding.
Body Section Roentgenography in Relation to Diagnosis in Diseases of the Nasal Accessory Sinuses. SHERWOOD MOORE, M.D., and ALFRED J. CONE, M.D., St. Louis.
Tumors of the Upper Jaw, Particularly in Relation to the Sinuses. CARL W. WALDRON, M.D., Minneapolis.
History and Present Status of Operations on the Labyrinthine Capsule for Otosclerosis. SAMUEL J. KOPETZKY, M.D., New York.

PROGRAMS FOR AFTERNOON SESSIONS

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

Tuesday, 2:00 p.m.—Stevens Hotel

ROBERT H. KENNEDY, M.D., New York; Chairman, Committee on Fractures and Other Traumas, Presiding.
End Results of Fractures of the Tibia and Fibula Treated by Skeletal Traction and Braun-Böhler Splint. HUBLEY R. OWEN, M.D., Philadelphia.
Fractures Due to Muscular Violence. FRANK P. STRICKLER, M.D., Louisville.
Undergraduate Education in Fractures and Other Traumas. CLAY RAY MURRAY, M.D., New York.
Extradural Hemorrhage Following Trauma. J. DERYL HART, M.D., and BARNES WOODHALL, M.D., Durham, N. C.
Fractures Involving the Ankle Joint. RALPH G. CAROTHERS, M.D., Cincinnati.

SYMPOSIUM ON CANCER

Wednesday, 2:00 p.m.—Stevens Hotel

FRANK E. ADAIR, M.D., New York; Chairman, Cancer Committee, Presiding.
Neurogenic Sarcoma. DONALD V. TRUEBLOOD, M.D., Seattle.
Present Status of Carcinogens and Hormones in Cancer Research. JOHN J. MORTON, M.D., Rochester, N.Y.
Radiation Damage to Tissue and Its Repair. ERNEST M. DALAND, M.D., Boston.
Pelvic Cancer. GEORGE KAMPERMAN, M.D., Detroit.

GROUP CLINICAL CONFERENCES

GENERAL SURGERY AND SURGICAL SPECIALTIES

Friday, 2:00 p.m.—Stevens Hotel

The Management of Uterine Prolapse. LOUIS E. PHANEUF, M.D., Boston, Presiding.
Deformities of the Ear, Congenital and Acquired. H. L. D. KIRKHAM, M.D., Houston, Presiding.
Major Trigeminal Neuralgia. FRANCIS C. GRANT, M.D., Philadelphia, Presiding.
Diagnosis and Treatment of Intrathoracic Tumors. FRANK S. DOLLEY, M.D., Los Angeles, Presiding.
Malignancies of the Kidney and Ureter. T. LEON HOWARD, M.D., Denver, Presiding.
Treatment of Compound Fractures. GUY A. CALDWELL, M.D., New Orleans, Presiding.

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Tuesday, 11:00 a.m.—Stevens Hotel

Surgical Indications in Glaucoma. HARRY S. GRADLE, M.D., Chicago, Presiding.
Rôle of Chemotherapy in Suppurative Diseases of the Middle Ear. JOHN J. SHEA, M.D., Memphis, Presiding.

Wednesday, 11:00 a.m.—Stevens Hotel

Surgical Management of Injuries to the Eye. ARTHUR M. CULLER, M.D., Dayton, Ohio, Presiding.
Indications for Surgery in Sinus Disease. SAMUEL IGLAUER, M.D., Cincinnati, Presiding.

Thursday, 11:00 a.m.—Stevens Hotel

Minor Surgery in Ophthalmology. MEYER WIENER, M.D., St. Louis, Presiding.
Osteomyelitis of the Skull Bones (Sinus and Mastoid Complications). THOMAS E. CARMODY, M.D., Denver, Presiding.

PANEL DISCUSSIONS

Monday, 1:30 to 3:00 p.m.—Stevens Hotel

Treatment of Acute Cholecystitis. JOHN L. ATLEE, JR., M.D., Lancaster, Pa., Presiding.

Collaborators: FREDERICK E. KREDEL, M.D., Charleston, S. C.; KENT W. BARBER, M.D., Quincy, Ill.; J. LOUIS RANSOHOFF, M.D., Cincinnati.

Dislocation of the Intervertebral Disc. FRANK D. DICKSON, M.D., Kansas City, Mo., Presiding.

Collaborators: W. JASON MIXTER, M.D., Boston; LLOYD NOLAND, M.D., Fairfield, Ala.; LEWIS J. POLLOCK, M.D., Chicago.

The Treatment of Endocrine Sterility in Woman. EDWIN C. HAMBLEN, M.D., Durham, N. C., Presiding.

Collaborators: JOHN C. BURCH, M.D., Nashville; CONRAD G. COLLINS, M.D., New Orleans.

Complications of Thyroid Surgery. FRANK H. LAHEY, M.D., Boston, Presiding.

Collaborators: HAROLD L. FOSS, M.D., Danville, Pa.; S. L. LEDBETTER, JR., M.D., Birmingham; H. H. SEARLS, M.D., San Francisco.

The Place of Irradiation Treatment in Cancer of the Breast. EUGENE P. PENDERGRASS, M.D., Philadelphia, Presiding.

Collaborators: FRANK E. ADAIR, M.D., New York; ALSON R. KILGORE, M.D., San Francisco; JAMES F. KELLY, M.D., Omaha; STUART W. HARRINGTON, M.D., Rochester, Minn.

Monday, 3:30 to 5:00 p.m.—Stevens Hotel

Abdominal Traumas. FREDERIC A. BESLEY, M.D., Waukegan, Presiding.

Collaborators: JOHN H. MULHOLLAND, M.D., New York; FRED W. BAILEY, M.D., St. Louis; AMBROSE H. STORCK, M.D., New Orleans.

Carcinoma of the Colon. FREDERICK A. COLLER, M.D., Ann Arbor, Mich., Presiding.

Collaborators: VERNON C. DAVID, M.D., Chicago; CLAUDE F. DIXON, M.D., Rochester, Minn.; THOMAS E. JONES, M.D., Cleveland.

Indications for Cesarean Section. S. A. COSGROVE, M.D., Jersey City, N. J., Presiding.

Collaborators: JAMES RAGLAN MILLER, M.D., Hartford; H. HUDNALL WARE, JR., M.D., Richmond.

Management of Acute Perforated Appendicitis. VERNE C. HUNT, M.D., Los Angeles, Presiding.

Collaborators: LAWRENCE S. FALLIS, M.D., Detroit; WILLIS D. GATCH, M.D., Indianapolis; HENRY K. RANSOM, M.D., Ann Arbor, Mich.

Subacromial Bursitis. HENRY W. MEYERDING, M.D., Rochester, Minn., Presiding.

Collaborator: EARL D. MCBRIDE, M.D., Oklahoma City.

Tuesday, 1:30 to 3:00 p.m.—Stevens Hotel

Atypical Giant Cell Tumors of Bone. BRADLEY L. COLEY, M.D., New York, Presiding.

Collaborator: DALLAS B. PHEMISTER, M.D., Chicago.

Nutritional State of the Patient (Vitamins, Proteins). L. KRAEER FERGUSON, M.D., Philadelphia, Presiding.

Collaborators: JOHN B. HARTZELL, M.D., Detroit; A. C. IVY, M.D., Chicago; CHARLES C. LUND, M.D., Boston.

Management of Infections of the Urinary Tract. CHARLES C. HIGGINS, M.D., Cleveland, Presiding.

Collaborators: HENRY O. MERTZ, M.D., Indianapolis; JOHN K. ORMOND, M.D., Detroit; IRA R. SISK, M.D., Madison, Wis.

Acute Abdominal Surgery in Children. THOMAS H. LANMAN, M.D., Boston, Presiding.

Collaborators: EDWARD J. DONOVAN, M.D., New York; WALTER ESTELL LEE, M.D., Philadelphia; W. J. POTTS, M.D., Oak Park, Ill.

Tumors of the Salivary Glands. ALBERT O. SINGLETON, M.D., Galveston, Presiding.

Collaborators: EDWIN I. BARTLETT, M.D., San Francisco; FREDERICK A. FIGI, M.D., Rochester, Minn.; R. B. MALCOLM, M.D., Chicago.

Tuesday, 3:30 to 5:00 p.m.—Stevens Hotel

Surgery in Pulmonary Tuberculosis. LEO ELOESSER, M.D., San Francisco, Presiding.

Collaborators: CASPER F. HEGNER, M.D., Denver; RICHARD H. MEADE, JR., M.D., Philadelphia; VICTOR STRONG RANDOLPH, M.D., Phoenix, Ariz.

Early Recognition of Cancer of the Uterine Cervix and of the Corpus. WILLIAM P. HEALY, M.D., New York, Presiding.

Collaborators: NORMAN F. MILLER, M.D., Ann Arbor, Mich.; FRED J. TAUSSIG, M.D., St. Louis; RAYMOND E. WATKINS, M.D., Portland, Ore.

Intestinal Obstruction. THOMAS G. ORR, M.D., Kansas City, Mo., Presiding.

Collaborators: M. J. HENRY, M.D., Louisville; CHARLES G. JOHNSTON, M.D., Detroit; MIMS GAGE, M.D., New Orleans.

Surgical Diseases of the Pancreas. ALLEN O. WHIPPLE, M.D., New York, Presiding.

Collaborators: ALEXANDER BRUNSCHWIG, M.D., Chicago; JOHN MILTON MCCAUGHAN, M.D., St. Louis.

Wednesday, 1:30 to 3:00 p.m.—Stevens Hotel

Tumors of the Urinary Bladder. HENRY G. BUGBEE, M.D., New York, Presiding.

Collaborators: LOUIS M. ORR, M.D., Orlando, Fla.; CARL RUSCHE, M.D., Los Angeles.

Etiology and Treatment of Gastric Hemorrhage. ROSCOE R. GRAHAM, M.D., Toronto, Presiding.

Collaborators: HOWARD M. CLUTE, M.D., Boston; J. WILLIAM HINTON, M.D., New York; J. SHELTON HORSLEY, M.D., Richmond.

Anesthesia in Cranial Surgery. GILBERT HORRAX, M.D., Boston, Presiding.

Collaborators: JOHN S. LUNDY, M.D., Rochester, Minn.; MAX MINOR PEET, M.D., Ann Arbor, Mich.

Healing of Wounds. SUMNER L. KOCH, M.D., Chicago, Presiding.

Collaborators: LAWRENCE CHAFFIN, M.D., Los Angeles; R. ARNOLD GRISWOLD, M.D., Louisville; EDWARD L. HOWES, M.D., Washington.

Liver Tests and Liver Function in Relation to Biliary Surgery. I. S. RAVDIN, M.D., Philadelphia, Presiding.

Collaborators: HENRY W. CAVE, M.D., New York; HOWARD K. GRAY, M.D., Rochester, Minn.; CHARLES B. PUESTOW, M.D., Chicago.

Wednesday, 3:30 to 5:00 p.m.—Stevens Hotel

Penetrating Wounds of the Brain. C. C. COLEMAN, M.D., Richmond, Presiding.

Collaborators: WALLACE B. HAMBY, M.D., Buffalo; HOWARD C. NAFFZIGER, M.D., San Francisco; BYRON STOOKEY, M.D., New York.

Infections of the Hand. MICHAEL L. MASON, M.D., Chicago, Presiding.

Collaborators: HOMER D. DUDLEY, M.D., Seattle; HENRY C. MARBLE, M.D., Boston; JAMES M. WINFIELD, M.D., Detroit.

Surgical Bacteriology. FRANK L. MELENEY, M.D., New York, Presiding.

Collaborators: EDMUND ANDREWS, M.D., Bloomington, Ill., J. DERYL HART, M.D., Durham, N. C.; WILLIAM A. ALTEMEIER, M.D., Cincinnati.

Immediate Treatment of Burns. GROVER C. PENBERTHY, M.D., Detroit, Presiding.

Collaborators: EDMUND BUTLER, M.D., San Francisco; HARRY C. HULL, M.D., Baltimore; DONALD B. WELLS, M.D., Hartford.

Anesthesia in Abdominal Surgery. ERWIN R. SCHMIDT, M.D., Madison, Wis., Presiding.

Collaborators: VIRGINIA APGAR, M.D., New York; CHARLES J. BETLACH, M.D., Chicago; F. W. HARTMAN, M.D., Detroit.

Thursday, 3:30 to 5:00 p.m.—Stevens Hotel

Anesthesia, Analgesia, and Amnesia in Obstetrics. LOUIS H. DOUGLASS, M.D., Baltimore, Presiding.
Collaborators: FREDERIC SCHREIBER, M.D., Detroit; PAUL TITUS, M.D., Pittsburgh; RALPH M. TOVELL, M.D., Hartford.

Non-malignant Diseases of the Intestines. THOMAS E. JONES, M.D., Cleveland, Presiding.
Collaborators: IRVIN ABELL, M.D., Louisville; CHARLES W. MAYO, M.D., Rochester, Minn.; J. L. MCGEHEE, M.D., Memphis.

Colles' Fracture. PAUL B. MAGNUSON, M.D., Chicago, Presiding.
Collaborators: ROBERT H. KENNEDY, M.D., New York; ROBERT D. SCHROCK, M.D., Omaha; GEORGE E. WILSON, M.D., Toronto.

Suppurative Conditions of the Lung. NORMAN S. SHENSTONE, M.D., Toronto, Presiding.
Collaborators: WILLIAM A. HUDSON, M.D., Detroit; WILLIAM F. RIENHOFF, JR., M.D., Baltimore; PAUL C. SAMSON, M.D., Oakland, Calif.

Management of Gangrene in Diabetics. ARTHUR A. ZIEROLD, M.D., Minneapolis, Presiding.
Collaborators: WALTER H. NADLER, M.D., Chicago; JOSEPH B. PRIESTLEY, M.D., Des Moines; BEVERLY CHEW SMITH, M.D., New York.

ASSEMBLY OF INITIATES

Monday, 3:00 p.m., Auditorium, American College of Surgeons, 40 East Erie St.

Processional—Initiates, Officers, Regents, and Governors.
Opening Remarks. GEORGE P. MULLER, M.D., Philadelphia, President.
The Program of the American College of Surgeons:
ARTHUR W. ALLEN, M.D., Boston, Vice Chairman, Board of Regents.
BOWMAN C. CROWELL, M.D., Associate Director.
MALCOLM T. MACEACHERN, M.D., Chicago, Associate Director.
The Fellowship Pledge. Recital by Initiates.
Greetings to the Initiates. EVARTS A. GRAHAM, M.D., St. Louis, President-elect.
Closing Remarks. IRVIN ABELL, M.D., Chairman, Board of Regents.
Signing of the Fellowship Roll. The Initiates.
Reception by the officers and regents for the fellows and initiates and members of their families.

ANNUAL HOSPITAL STANDARDIZATION CONFERENCE

Monday, 10:00 a.m.—Ballroom, Stevens Hotel

GEORGE P. MULLER, M.D., Philadelphia; President, American College of Surgeons, presiding.
Address of the President—The Hospital Program of the American College of Surgeons.
Report of the 1940 Hospital Standardization Survey—Official Announcement of the List of Approved Hospitals. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents, American College of Surgeons.
Report on Graduate Training in Surgery. DALLAS B. PHEMISTER, M.D., Chicago.
The Hospital of Tomorrow. REV. A. M. SCHWITALLA, S.J., St. Louis; President, Catholic Hospital Association.
Medical Preparedness for National Emergency. ROSS T. MCINTIRE, M.D., Washington; Surgeon-General, United States Navy.
Preparedness for National Emergency from the Standpoint of the Hospital. E. W. JONES, Albany, N. Y.
The Effect of the Present Trend in Specialization in Medicine on Hospital Administration and Service. BENJAMIN W. BLACK, M.D., Oakland, Calif.; President, American Hospital Association.

Monday, 2:00 p.m.—Ballroom, Stevens Hotel

HARRY E. MOCK, M.D., Chicago, presiding.
Convalescent Care—The Missing Link in the Cure of Many Patients. HARRY E. MOCK, M.D., Chicago.
Socio-Economic Aspects of Convalescent Care. ELIZABETH G. GARDINER, New York.
Minimum Standards for Convalescent Hospitals. E. H. L. CORWIN, Ph.D., New York.
Essentials in Planning of Institutions for Convalescent Care. WILLIAM H. WALSH, M.D., Chicago.
Institutional Convalescent Care for Various Types of Patients:
- The Surgical Patient. W. EDWARD GALLIE, M.D., Toronto.
- The Medical Patient. NEWELL C. GILBERT, M.D., Chicago.
- The Cancer Patient. FRANK E. ADAIR, M.D., New York.
- The Fracture Patient. ROBERT H. KENNEDY, M.D., New York.

General discussion. Opened by KARL A. MEYER, M.D., Chicago; JOHN S. COULTER, M.D., Chicago; JOSEPH KING, Chicago.

Monday 4:30 p.m.—Lobby, 3rd Floor, Stevens Hotel

Consultation Service.

Tuesday, 7:45 a.m.—Private Dining Room, Stevens Hotel

Breakfast Conference, Sponsored by the American College of Hospital Administrators in co-operation with the American College of Surgeons: The Hospital Administrators' Program for Self Development. ARTHUR C. BACHMEYER, M.D., Chicago, presiding.
Discussion from the following aspects:
- Institutes for Hospital Administrators. BENJAMIN W. BLACK, M.D., Oakland, Calif.
- Apprenticeship Training. CLAUDE W. MUNGER, M.D., New York.
- Self Study and Personal Development. JOSEPH G. NORBY, Milwaukee.

General discussion. Opened by FLORENCE KING, St. Louis.

Tuesday, 9:30 a.m.—Upper Tower Ballroom, Stevens Hotel

GUY M. HANNER, Colorado Springs, Colo., presiding.
Greetings from the American Protestant Hospital Association. GUY M. HANNER, Colorado Springs.
Panel Discussions:
- Qualifications for Performing Major Surgery. Conducted by HAROLD L. FOSS, M.D., Danville, Pa.
- Discussion by CLARENCE E. REESE, M.D., San Diego, Calif.
- The Respective Rôles of the Professional and Administrative Staffs in the Control of Postoperative Infections. Conducted by FRANK L. MELENEY, M.D., New York.
  - Operating Room Supervisor. MARGARET K. SCHAFER, Ann Arbor, Mich.
- Anesthesia Hazards. Conducted by JOHN S. LUNDY, M.D., Rochester, Minn.
  - Physical Hazards—Explosibility and Inflammability. RALPH M. TOVELL, M.D., Hartford.
  - Administrative Hazards—The Administration of Anesthesia by Inexperienced Persons. HENRY S. RUTH, M.D., Merion, Pa.

Tuesday, 2:00 p.m.—Upper Tower Ballroom, Stevens Hotel

Conference on Nursing Service and Nursing Education. NELLIE X. HAWKINSON, R.N., Chicago, presiding.
Essentials of Good Nursing Care of the Patient. SISTER M. BERENICE, R.N., Milwaukee.
Panel Discussions:
- Administrative Costs of Nursing Based on Recent Study and Published Report. Conducted by CLAUDE W. MUNGER, M.D., New York.
- Discussion from the viewpoints of:
  - Special Problems in the Separation of Nursing Education and Nursing Costs. CHARLES A. ROVETTA, Chicago.
  - The Responsibility and Contribution of the Nursing Administrator in a Nursing Cost Study. BLANCHE PFEFFERKORN, R.N., New York.
  - Accounting in Nursing Costs. C. RUFUS REREM, Ph.D., Chicago.
- General Discussion.
- The General Plan for Accrediting Schools of Nursing. Conducted by ELIZABETH C. BURGESS, R.N., New York.
- Discussion from the viewpoints of:
  - Consultant to Committee on Accrediting, National League of Nursing Education. GEORGE A. WORKS, Chicago.
  - Hospital Administrator. ROBIN C. BUERKI, M.D., Madison.
  - Nursing Administrator. MARGARET CARRINGTON, R.N., Chicago.
- General Discussion.

Tuesday, 4:30 p.m.—Lobby, 3rd Floor, Stevens Hotel

Consultation Service.

Tuesday, 7:30 p.m.—Upper Tower Ballroom, Stevens Hotel

Panel Round Table Conference: Organization, Management, and Special Problems of the Small Hospital. A. F. BRANTON, M.D., Willmar, Minn., presiding.

Personnel—Securing and Maintaining Competent Administrative and Other Personnel. MARJORIE M. IBSEN, R.N., Highland Park, Ill.

Medical Staff—Organizing and Functioning of the Medical Staff. F. JANE GRAVES, R.N., Alton, Ill.

Adjunct Services—Providing Adequate Laboratory, X-ray, and Other Essential Services for the Treatment of the Patient. F. P. MCNAMARA, M.D., Dubuque, Iowa.

Medical Records—Securing Good Medical Records. ALPHONSUS M. MCCARTHY, M.D., Electric Mills, Miss.

Nursing—Providing Efficient Nursing Service. MABEL L. KUEBLER, R.N., Louisville.

Financing—Meeting the Problem of Finance. JEWELL W. THRASHER, R.N., Dothan, Ala.

The Women's Auxiliary—Organization, Management, and Special Problems. ANNE F. GOODYEAR, Hastings, Mich.

Public Relations—Promoting a Public Relations Program. CHARLOTTE GARRISON, R.N., Battle Creek, Mich.

Hospital Standardization—Meeting the Requirements for Approval by the American College of Surgeons. E. W. WILLIAMSON, M.D., Chicago.

Collaborators: GRAHAM L. DAVIS, Battle Creek, Mich.; PAUL H. FESLER, Nopeming, Minn.

Wednesday, 7:45 a.m.—Private Dining Room, Stevens Hotel

Breakfast Conference, Sponsored by the American Association of Medical Record Librarians in co-operation with the American College of Surgeons. The Medical Records Librarians' Program for Self Development.

Discussion from the following aspects:

Approved Courses for Medical Records Librarians. ALICE G. KIRKLAND, Oakland, Calif.

Need for Courses for Medical Records Librarians Already in the Field. EDNA K. HUFFMAN, Chicago.

Self Study and Personal Development. MARGARET E. HOOVLER, Pittsburgh.

General Discussion.

Wednesday, 9:30 a.m.—Upper Tower Ballroom, Stevens Hotel

Joint Conference with American Association of Medical Record Librarians. ROBIN C. BUERKI, M.D., Madison, presiding.

Greetings from the American College of Surgeons to the American Association of Medical Record Librarians. MALCOLM T. MACEACHERN, M.D., Chicago.

Personal, Professional, and Other Qualifications of a Successful Medical Records Librarian. DOROTHEA M. TROTTER, R.R.L., Grand Rapids, Mich.

The Adoption and Putting Into Operation of the Standard Medical Nomenclature. EDWIN P. JORDAN, M.D., Chicago.

Surgical Nomenclature. HILGER P. JENKINS, M.D., Chicago.

The Practical Application of the Punch Card System in Assembling Statistical and Research Data from the Medical Records. JESSIE L. HARNED, R.R.L., Durham, N. C.

Guiding Principles of the Medical Records Librarian in Her Relations with the Resident Staff. SISTER MARY SERVATIA, R.R.L., St. Louis.

Open Forum Discussion: Problems Pertaining to the Obtaining of Good Medical Records. Conducted by ROBIN C. BUERKI, M.D., Madison, Wis.

Wednesday, 12:00 m.—Lobby, 3rd Floor, Stevens Hotel

Consultation Service.

Wednesday, 2:30 p.m.—Local Hospitals

Group Conferences and Demonstrations.

Augustana Hospital. E. I. ERICKSON, Superintendent.

Central Service for Surgical Dressings. AGNES HANSON, R.N.

Rehabilitation and Modernization of the X-ray Department. DAVID S. BEILIN, M.D.

Management of the Pharmacy Service. FRANCES DOUGLAS, Ph.G.

Children's Memorial Hospital. MABEL W. BINNER, R.N., Superintendent.

Outpatient Clinic Management; Inspection of New Clinic Building. BABETTE S. JENNINGS.

Organization and Management of Laundry and Linen Service. ALOIS J. ULRICH.

Purchasing and Stores. CARRIE H. JONES.

Cook County Hospital. GENERAL MANUS MCCLOSKEY, Warden.

Central Control of Parenteral Solutions. FREDERICK STEIGMANN, M.D.

Preservation and Use of Medical Records in a Large Hospital. STELLA FORD WALKER.

Organization and Management of a Blood Bank Service. ELIZABETH SCHIRMER, M.D.

Grant Hospital. CLINTON F. SMITH, Administrator.

Central Stores; Perpetual Inventory; Purchasing. ROLAND A. SCOTT.

Central Service. MARGARET KARSTEN MIHULA.

Medical Records Librarians' School. EDNA K. HUFFMAN.

Department of Nursing: Conference Featuring Ward Instruction and Ward Supervision. ELIZABETH C. WIVEL, R.N.

Henrotin Hospital. VERONICA MILLER, R.N., Administrator.

Office Management. HENRIETTA DRAGER.

Central Control of Supplies. MILDRED KRAUSE, R.N.

Housekeeping. MILDRED PAGE.

Michael Reese Hospital. ALBERT H. SCHEIDT, Associate Director.

Personnel Management. GEORGE PECK.

Clinic Management and Medical Social Service. ELIZABETH MCCONNELL and LEONORA RUBINOW.

Modern Laundry Methods. R. J. GABRIELSON.

Presbyterian Hospital. ASA S. BACON, Superintendent.

Personality in the hospital—the Front Office—Contact and Dealing with Patients, Relatives, and Friends. ASA S. BACON.

Business Methods—Accounting, Purchasing, Income, Charges, Control. HERMAN HENSEL and LESLIE D. REID.

Centralized Food Service. BEULAH HUNZICKER.

St. Joseph Hospital. SISTER ZITA, A.M., Administrator.

Organization and Management of the Obstetrical Department: Rehabilitation of the Physical Plant. CLYDE GEIGER, M.D., and EMILY MARCOULIER.

Operating Room Management, Technique and Procedures; Rehabilitation of the Physical Plant. SISTER DELPHINE, R.N., B.S.

Food Administration. SISTER RUFINA, R.N., B.S.

University of Chicago Clinics. G. OTIS WHITECOTTON, M.D., Superintendent.

Anesthesia; Operating Room Management and Technique. HUBERTA LIVINGSTONE, M.D., and DOROTHY SCHMIDT, R.N.
Food Service. ELLA M. ECK.
Medical Records Library; Creation of a Medical Record. ADALINE HAYDEN.
Women and Children's Hospital. EDNA H. NELSON, Superintendent.
Mothers' Milk Bureau. MARION GREENE.
Care of Newborn. MARIE HARLAN.
Obstetrical Technique and Procedures. BEATRICE E. TUCKER, M.D.

Wednesday, 7:30 p.m.—Upper Tower Ballroom, Stevens Hotel

Departmental Panel Round Table Conference. Conducted by ROBIN C. BUERKI, M.D., Madison, Wis., and MALCOLM T. MACEACHERN, M.D., Chicago.
Administrative Practices. THOMAS T. MURRAY, Albany.
Anesthesia Service. HUGH A. CUNNINGHAM, M.D., Milwaukee.
Patients' Libraries. SELMA LINDEM, Chicago.
Business Management. WILLIS J. GRAY, Detroit.
Food Service. HELEN MAE BRYAN, Chicago.
Laundry and Linen Service. ALOIS J. ULRICH, Chicago.
Medical Records. MARGARET C. TAYLOR, Rochester, N. Y.
Medical Social Service. JOSEPHINE G. TAYLOR, Chicago.
Medical Staff. CHARLES B. PUESTOW, M.D., Chicago.
Nursing Service. ELIZABETH ODELL, Evanston, Ill.
Obstetrical Service. CARL P. HUBER, M.D., Indianapolis.
Outpatient Department. ALMA VAUPEL, Milwaukee.
Pathological Service. LALL G. MONTGOMERY, M.D., Muncie, Ind.
Physical Therapy. JOHN S. COULTER, M.D., Chicago.
Tuberculosis. M. POLLAK, M.D., Peoria, Ill.
Utilities. OSCAR E. OLSON, Madison, Wis.
X-ray Service. JAMES T. CASE, M.D., Chicago.

Thursday, 7:45 a.m.—Private Dining Room, Stevens Hotel

Breakfast Conference. The Public Relations Program of Your Hospital. ALDEN B. MILLS, Chicago, presiding.
Discussion from the following aspects:
Newspaper Publicity. LAURA JACKSON, Chicago.
Annual Reports. ASA S. BACON, Chicago.
Syndicated Material. ALBERT G. HAHN, Evansville, Ind.
Fund Raising. ADA BELLE MCCLEERY, R.N., Evanston, Ill.
General Discussion. Opened by CARL I. FLATH, Detroit.

Thursday, 9:30 a.m.—Upper Tower Ballroom, Stevens Hotel

Panel Discussion on Hospital Problems of Current Interest to the Hospital. FRANK R. BRADLEY, M.D., St. Louis, presiding.
Hospital Administration and Its Relation to Organized Medicine. BORIS FINGERHOOD, Brooklyn.
The Problem of Tuberculosis in the General Hospital. ERNEST S. MARIETTE, M.D., Oak Terrace, Minn.
An Organized Health Service for Hospital Employees. ROBERT E. NEFF, Iowa City, Iowa.
The Implications of Good Medical Staff Organization. JOSEPH G. NORBY, Milwaukee.
Hospital Rates. ALBERT H. SCHEIDT, Chicago.

Thursday, 12:00 m.—Lobby, 3rd Floor, Stevens Hotel

Consultation Service.

Thursday, 2:30 p.m.—Local Hospitals

Group Conferences and Demonstrations.

Chicago Lying-In Hospital and Dispensary. S. A. FERGUSON, Assistant to Director.
Registration and Admission. JEAN B. ANDERSON.
Isolation of Obstetrical Patients. GEORGIA HUKILL, R.N.
Nursery Management. GEORGIA HUKILL, R.N.
Mount Sinai Hospital. STEPHEN MANHEIMER, M.D., Director.
Medical Staff Organization and Administrative Control. STEPHEN MANHEIMER, M.D.
Clinical Laboratory—Organization, Management and Service. I. DAVIDSOHN, M.D.
Business Management—Accounting, Purchasing and Revenue. NATHAN W. HELMAN, C.P.A.
Passavant Memorial Hospital. IRVING S. CUTTER, M.D., Superintendent.
Oxygen Therapy. M. HERBERT BARKER, M.D.
Physical Therapy. JOHN S. COULTER, M.D.
Engineering and Maintenance Problems. WILLIAM W. DAVISON.
Ravenswood Hospital. J. DEWEY LUTES, Administrator.
General Administration of a 200 Bed Hospital. J. DEWEY LUTES.
Medical Records Department. HELENA BENDER.
Medical Library. LEONORA PERRY.
Research and Educational Hospitals. MAJOR H. WORTHINGTON, M.D., Managing Officer.
Psychiatry in a General Hospital; Inspection of New Neuro-Psychiatric Building. I. R. SONENTHAL, M.D.
Central Supply Service and Central Linen Exchange. ANN LUCILLE LAIRD.
Study Tour of Orthopedic Hospital. ANTOINETTE J. RADEK, R.N., Assistant Chief Nurse.
St. Elizabeth Hospital. SISTER M. ADOLPHINE, R.N., Superior.
Obstetrical Department in a General Hospital. J. R. LAVIERI, M.D.
Central Supply Service. SISTER M. MARGARITIS, R.N.
Modernized Nursery Technique. MARIE DALTON, R.N.
St. Luke's Hospital. CHARLES A. WORDELL, Director.
Business Methods—Accounting, Purchasing, Stores, Preparation of Budget, Monthly Reports. FREDERICK T. MUNCIE.
Equipment and Procedures for Dressing Cart, Modified Wangensteen, or Gravity Suction. EVELYN VAN DE STEEG.
Set-Up in Major Operating Rooms with Special Reference to Personnel; Blood Transfusions. MARIE ANTE.
Wesley Memorial Hospital. E. R. SNYDER, Superintendent.
Practical Problems in Anesthesia and Oxygen Therapy. MARY KARP, M.D.
Food Service. ELIZABETH H. TUFT.
Procedures in the Obstetrical Department. VERTE J. JOHNSON.
West Suburban Hospital. L. C. VONDER HEIDT, Superintendent.
Management of the Newborn; Humidity a Dominant Nursery Safeguard. LEONORA SCHULZE, R.N., MARY MOLLEY, R.N., and L. C. VONDER HEIDT.
Palmprint Identification. GILBERT P. POND, M.D.
Maintenance of the Physical Plant and Engineering. DAVID PATTERSON.
Woodlawn Hospital. GRACE L. DEVILBISS, R.N., Superintendent.
A Study of Organization, Management and Procedures in a Private Hospital of 100 Beds. GRACE L. DEVILBISS, R.N., and Staff.

PRELIMINARY CLINICAL PROGRAM

ARRANGED IN THE FOLLOWING SUBDIVISIONS: GENERAL SURGERY, OBSTETRICS AND GYNECOLOGY, GENITO-URINARY SURGERY, FRACTURES AND OTHER TRAUMAS, ORTHOPEDIC SURGERY, THORACIC SURGERY, NEUROSURGERY, OPHTHALMOLOGY, OTOLARYNGOLOGY.

GENERAL SURGERY

Monday

CHICAGO MEMORIAL HOSPITAL

PETER S. CLARK, CHARLES J. DRUECK, ROBERT A. MELENDY, BENNETT R. PARKER, JOHN VAN PROHASKA, M. L. WEINSTEIN, LEO M. ZIMMERMAN, and GEORGE M. LANDAU—1. Operative and dry clinic:

JOHN VAN PROHASKA. Surgical aspects of carcinoma of ampulla of Vater; enterogenous cysts in infants.

M. L. WEINSTEIN. Resection of stomach with DePetz instrument; rectal anesthesia.

LEO M. ZIMMERMAN. Essential problems in the surgical treatment of inguinal hernia.

BENNETT R. PARKER. Problems in gall-bladder surgery.

COOK COUNTY HOSPITAL

Staff—2. Symposium on biliary tract disease.

EDMOND F. FOLEY. Medical aspects.

HANS L. POPPER. Perverted physiology.

FREDERICK STEIGMAN. Jaundice.

HARRY CULVER. Urological factors in differential diagnosis.

WALTER SCHILLER. Pathology.

M. J. HUBENY. Roentgenological aspects in diagnosis.

LINDON SEED. Clinical results with synthetic Vitamin K in jaundice.

RAYMOND W. MCNEALY. Pre- and postoperative management.

KARL A. MEYER. Operative management.

MARSHALL DAVISON. Post-cholecystectomy pain.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

MICHAEL REESE HOSPITAL

Staff—2. Symposium on transfusion and infusion.

LEO M. ZIMMERMAN. History of blood transfusion.

SIDNEY O. LEVINSON. General survey of a modern transfusion department.

HAROLD LAUFMAN. Blood transfusion reactions, statistical study.

ANNA MARIE STRAUSS. Experiences with indirect transfusion reactions.

H. NECHELES. Plasma and serum as substitutes for whole blood transfusions; physiology and laboratory experiments.

FRANK RUBOVITS. Clinical experience with plasma and serum transfusions.

Inspection of transfusion and serum departments.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Cancer of the large bowel and appendicitis.

Tuesday

AUGUSTANA HOSPITAL

NELSON M. PERCY and OSCAR E. NADEAU—9. Operative and dry clinics.

ALBERT MERRITT BILLINGS HOSPITAL

L. R. DRAGSTEDT and HILGER P. JENKINS—9. Operative and dry clinic.

COOK COUNTY HOSPITAL

KARL A. MEYER—9. Operative clinic: Stomach and colon.

JOHN R. HARGER—9. Operative clinic: Peridural anesthesia for major operations.

C. C. GUY—9. Dry clinic: Surgical problems in the diabetic.

SUMNER L. KOCH—9. Dry clinic: Injuries and infections of the hand.

RAYMOND W. MCNEALY—10. Operative clinic: Gallbladder; gastric surgery.

E. M. MILLER—10. Dry clinic: Bowel obstruction in the newborn; gangrene complicating scarlet fever; volvulus of sigmoid colon in children.

J. D. KOUCKY—10. Operative and dry clinic: Arteriosclerotic gangrene of the legs.

JOHN B. O'DONOGHUE—10. Operative clinic: Gastrointestinal.

Staff—2. Symposium on military organization in the care of wounded under war conditions.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

Staff—1. Demonstration on cadaver, some principles in gall-bladder surgery.

EVANGELICAL HOSPITAL

P. E. HOPKINS, G. E. JOHNSON, and JAMES PATEJDL—9. Operations.

EVANSTON HOSPITAL

W. R. PARKES, FREDERICK CHRISTOPHER, W. KENNETH JENNINGS, J. PEERMAN NESSELROD, JAMES P. GRIER, J. E. KEARNS, and FREDERICK WILLIAM MERRIFIELD—9. Operative and dry clinic:

FREDERICK CHRISTOPHER. Abdomino-perineal resection for carcinoma of the rectum.

W. KENNETH JENNINGS. The colostomy.

JAMES P. GRIER. Surgery of the biliary tract.

J. E. KEARNS. Vascular clinic.

J. PEERMAN NESSELROD. Motion pictures of the rectum.

FREDERICK W. MERRIFIELD. Oral surgery.

GRANT HOSPITAL

KARL A. MEYER—9. Operations.
A. G. ZIMMERMAN—9. Mediastinal tumors.

MICHAEL REESE HOSPITAL

Staff—9. Operative clinic:
JAMES PATEJDL. Gastro-intestinal.
R. B. BETTMAN and WILLIAM TANNENBAUM. Gallbladder.
A. A. STRAUSS and S. F. STRAUSS. Stomach and colon.
S. L. GOLDBERG. General.
Staff—9. Dry clinic: Symposium on peptic ulcer.
H. NECHELES. Newer conceptions of the physiology of stomach in relation to ulcer.
JACOB MEYER. The bleeding ulcer.
H. F. BINSWANGER. Treatment of bleeding ulcer with colloidal preparations.
S. F. STRAUSS. Indications for gastric surgery.
A. A. STRAUSS. Subtotal gastric resection.
JAMES PATEJDL. New methods in gastric surgery.
Staff—2. Dry clinic:
LEO M. ZIMMERMAN. Anatomy of inguinal hernia in relation to surgery.
NATHAN N. CROHN. Incidence of strangulation in inguinal hernia; treatment of hernia.
LEO M. ZIMMERMAN and H. LAUFMAN. Unusual complications of inguinal hernia.
R. B. BETTMAN and WILLIAM TANNENBAUM. Wound evisceration.
S. L. GOLDBERG. Septicemia.
D. C. STRAUSS. Epiphyseal separations.
Staff—2. Dry clinic: Peripheral vascular surgery.
LEO M. ZIMMERMAN and S. MUSCOVITZ. Thrombophlebitis and pulmonary embolism.
H. A. ROTH. Indications and technique of saphenous vein ligation.
SAMUEL PERLOW and S. S. HALPERN. Prostigmin in peripheral vascular disease.
SAMUEL PERLOW. Injection treatment of angina pectoris; sympathectomy for peripheral vascular disturbances.
S. STAGMAN and E. BLUMENTHAL. Treatment of varicose veins in the aged.
SAMUEL PERLOW and E. BLUMENTHAL. Ligation of the lesser saphenous vein.

RAVENSWOOD HOSPITAL

Staff—9. Operations.
Staff—10. Tumor clinic: Scientific exhibit; demonstration of procedure.
Staff—10. End-results of varicose vein operations.

RESEARCH AND EDUCATIONAL HOSPITALS

GÉZA DE TAKATS, J. T. REYNOLDS and staff—9. Operative clinic and symposium on hypertension: Splanchnic nerve section for hypertension.
S. R. ROSENTHAL. Renal biopsies.
F. K. HICK. Physiology of the heart in hypertension.
H. C. LUETH. The kidney function in hypertensive states.
H. E. HEYER. The brain in hypertensive states.
R. O. RISER. The eye grounds in hypertension—illustrated by kodachrome slides.
GÉZA DE TAKATS and associates. The surgical approach to the problem of hypertension.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. JOSEPH HOSPITAL

Staff—9. Operative and dry clinics:
HUGH MCKENNA. Cholecystectomy; thyroidectomy; closure of colon following second-stage miculiz operation for carcinoma of descending colon.
WILLIAM BECK. Acute intestinal obstruction.
BERT FITZGERALD. Thyroidectomy.
LEONARD KRATZ. Ruptured appendicitis.
FRANK MCCARTY. Thyroidectomy; cholecystectomy; stomach surgery; gall-bladder disease.
J. A. WIMBERLY. Thyroidectomy.
STEPHEN A. ZEIMAN. Herniotomy; moving pictures of hernia operation.

ST. LUKE'S HOSPITAL

W. R. CUBBINS—9. Operations.
SELIM W. MCARTHUR—9. Operations.
Staff—9. Dry clinic:
E. C. HOLMBLAD and R. A. JACOBSON. Treatment of gas gangrene.
T. L. HANSEN and J. M. L. JENSEN. Shelf fractures of tibia.
JOHN D. ELLIS. Amputations.
JOHN L. LINDQUIST. Physiological problems and management in postoperative gastro-intestinal constant suction drainage.
G. V. PONTIUS. Hypertrophic pyloric stenosis in the adult.
H. I. MEYER. Paget's disease; osteitis deformans.
W. R. CUBBINS. Combined abdominal and chest injuries with differential diagnosis.
ERIC OLDBERG. Spinal injuries.
ALFRED P. SOLOMON. Personality disorders during retarded convalescence following industrial injuries.
H. E. MOCK and associates—9. Skull fracture exhibit.
PAUL W. GREELEY—10. Operations: Total reconstruction of external ear; rhinoplasty.

WESLEY MEMORIAL HOSPITAL

RAYMOND W. MCNEALY—9. Thyroidectomy; resection of colon.
S. J. FOGELSON—9. Gastric resection.
WILLIAM M. MCMILLAN—9. Repair of inguinal hernia.
JOSEPH E. SCHAEFER and KENNETH W. PENHALE—9. Repair of cleft palate.
WILLIAM A. HENDRICKS—9. Demonstrations: Lymphosarcoma of the stomach; stab wound of the heart with repair; isolated simple non-specific ulcer of the cecum, healing without surgery.
GUY S. VAN ALSTYNE—9. Demonstration: Common duct stone with rupture of common duct.
EARL O. LATIMER—10. Gastric resection.
WILLIAM MILLER—10. Cholecystectomy.
NORMAN G. PARRY—10. Cholecystectomy.

MUNICIPAL TUBERCULOSIS SANITARIUM

CLEMENT L. MARTIN—9. Anorectal tuberculosis.

Wednesday

AUGUSTANA HOSPITAL

A. T. LUNDGREN and EARL GARSIDE—9. Operative and dry clinic.
JOHN W. NUZUM and RUDOLPH ODEN—9. Operative and dry clinic.

CHICAGO MEMORIAL HOSPITAL

Medical, surgical x-ray and pathological departments—2. Symposia: Treatment of pneumonia with serum and sulfapyridine; peptic ulcer.

CHILDREN'S MEMORIAL HOSPITAL

ALBERT H. MONTGOMERY, JOHN A. GRAHAM, JAY IRELAND, W. J. POTTS, J. J. MUSSIL, A. E. DIGGS, and S. E. LAWTON—9. Operations.

COOK COUNTY HOSPITAL

VICTOR L. SCHRAGER—9. Operations.
RALPH C. SULLIVAN—9. Operations.
EDWARD A. CHRISTOFFERSON—9. Operative clinic: Appendicitis.
FRANK J. JIRKA—10. Dry clinic: Dysgerminoma in the male.
J. R. BUCHBINDER—1. Operative clinic: Thyroid; biliary tract.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

Staff—1. Demonstrations on cadaver and anesthetized dog. Gastric surgery.

HENROTIN HOSPITAL

ROBERT F. SHARER—9. Operative and dry clinic: Gastrojejunal ulcers.

JACKSON PARK HOSPITAL

C. C. CLARK—9. Abdominal operations.
A. BAMBERGER—10. Abdominal and thyroid operations.
H. H. COX—10. Operative and dry clinic.

LORETTO HOSPITAL

E. PERRY VAUGHN—9. Operations.
ALLEN E. STEWART and L. NEWMAN—10. Operative and dry clinic.
FRANK M. SYLVESTER—10. Operations.
LOUIS F. PLZAK and FRANK F. KOTALIK—10. Goiter clinic.

MERCY HOSPITAL

M. F. MCGUIRE—9. Operative and dry clinic: Biliary tract.
WILLIAM J. PICKETT—9. Operative and dry clinic: Gastric surgery.
C. F. SAWYER—9. Intestinal obstruction.
J. SANDROY—9. Tests for liver function.

MICHAEL REESE HOSPITAL

Staff—9. Operative clinic:
M. L. PARKER. Thyroid.
A. A. STRAUSS and S. F. STRAUSS. Gastro-intestinal.
R. B. BETTMAN and WILLIAM TANNENBAUM. Surgery of extra hepatic bile tracts.
NATHAN N. CROHN. Subject to be announced.
LEO M. ZIMMERMAN. Hernia.
Staff—9. Dry clinic: Symposium on diseases of the bile tracts.
H. NECHELES. New knowledge of physiology of the gall-bladder.
S. PORTIS. Medical indications for surgery of the gall-bladder.
A. M. SERBY. Liver function tests.
R. B. BETTMAN. Technique of gall-bladder operations with motion pictures.
WILLIAM TANNENBAUM. Carcinoma of the gall-bladder.
G. M. LICHTENSTEIN. End-results of gall-bladder surgery.
NATHAN N. CROHN. Measurements of the common and cystic ducts.
ROBERT ARENS and R. B. BETTMAN. Immediate cholangiography.
Staff—2. Dry clinic:
LEON BLOCH. Ulcerative colitis.
A. A. STRAUSS. Indications for ileostomy in colon surgery.
S. F. STRAUSS. Surgical lesions of the colon.
ROBERT ARENS. X-ray diagnosis of diseases of the colon.
OTTO SAPHIR. Pathology of the colon.
H. NECHELES. Newer physiology of the intestines.

MOTHER CABRINI MEMORIAL HOSPITAL

E. J. CHESROW and associates—9. Abdominal operations.

PASSAVANT MEMORIAL HOSPITAL

SUMNER L. KOCH, MICHAEL L. MASON, and HARVEY ALLEN—9. Dry clinic: Presentation of cases illustrating problems in surgery of the hand; discussion of wound infections.

RESEARCH AND EDUCATIONAL HOSPITALS

WARREN H. COLE and staff—9. Operative clinic and thyroid symposium: Thyroidectomy; prerequisites for thyroidectomy.
R. B. MALCOLM. Surgical pathology of goiter.
J. T. REYNOLDS. Postoperative treatment.
CHARLES B. PUESTOW. Suture material and end-results.
LINDON SEED. King operation for recurrent nerve paralysis.
R. W. KEETON. Management of patients with hyperthyroidism and cardiac disease.
P. W. GREELEY—9. Thick razor graft to axillary contracture; island artery flap to nose; costal cartilage graft to nose; full thickness skin graft to hand; demonstration of finished plastic cases.
P. J. SARMA—9:30. Varicose vein clinic.
WARREN H. COLE—11:15. Splenectomy.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. ANNE'S HOSPITAL

G. F. THOMPSON, J. J. GEARIN, J. L. KNAPP, J. W. KEANE, ADOLPH KRAFT, and H. M. PETERSON—9. Operations.
ADOLPH KRAFT—1. Dry clinic: Carcinoma of cecum.
G. F. THOMPSON—1. Dry clinic: Carcinoma of breast; blastomycosis of cecum.

ST. BERNARD'S HOSPITAL

WILLIAM G. EPSTEIN—9. Thyroid operations.
L. B. DONKLE, G. M. CUSHING, and WILLIAM MULHOLLAND—9. Abdominal operations.
S. L. GOVERNALE—9. Dry clinic: Pseudohypertrophic muscular dystrophy.

ST. LUKE'S HOSPITAL

H. E. MOCK—9. Operations.
Staff—9. Dry clinic:
SELIM W. MCARTHUR. Stomach deformities simulating malignancy in x-ray study.
F. L. MCMILLAN. Lesions of the colon.
H. E. JONES. Treatment of complete biliary fistulas.
E. LEE STROHL. Common duct obstructions.
W. G. DIFFENBAUGH. Autoplastic operation for hernial repair; statistical report.
H. E. MOCK. Breast tumors; malignancies and granulomas of the gastro-intestinal tract.
E. W. RYERSON. Conservative treatment of fractures.
T. P. GRAUER. Injuries of the genito-urinary tract.
HAROLD A. SOFIELD. Fractures of femoral neck.
H. E. MOCK and associates—9. Skull fracture exhibit.

SWEDISH COVENANT HOSPITAL

R. F. ELMER, W. B. STROMBERG, and A. C. PETERSON—9. Operative clinic: Surgery of stomach and gall tracts.
K. L. VEHE, A. P. SANDAHL, and R. E. TALBOTT—9. Head and neck infections; anatomical demonstrations.
M. R. BROMAN—9. Problems in pathology in a private hospital.
R. G. WILLY—9. Diagnostic problems studied in a private hospital.

VETERANS ADMINISTRATION FACILITY

P. F. BROWN and B. F. WARD—9. Abdominal operations.
R. B. MORELAND—9. Operative clinic: Tumors.
A. E. WILLIAMS—9. Inspection tour of deep x-ray and radium unit.
G. R. ALLABEN—10. Dry clinic: Tumor clinic in the general hospital; method of presentation of cases before Tumor Board; diagnosis, treatment, and follow-up.
MAX CUTLER—11. Dry clinic: Carcinoma of the mouth and larynx; problems in diagnosis and treatment; indications for surgery and irradiation in laryngeal carcinoma; presentation of cases.

Thursday

ALEXIAN BROTHERS' HOSPITAL

WILLIAM J. SWIFT—9. Herniotomy.
DANIEL E. MURPHY—9. Circulatory disturbances of lower extremity.
FREDERICK A. RETTIG—10. Abdominal surgery and postoperative care.

AUGUSTANA HOSPITAL

NELSON M. PERCY and OSCAR E. NADEAU—9. Operative and dry clinics. Symposium on diagnosis and surgery of goiter.

CHICAGO MEMORIAL HOSPITAL

CASPER M. EPSTEIN—9. Operative clinic: Correction of cleft palate; facial plastic.

CHILDREN'S MEMORIAL HOSPITAL

ALBERT H. MONTGOMERY, JAY IRELAND, and J. J. MUSSIL—2:30. Demonstration of cases: Abdominal tumors in children; subphrenic abscess, osteomyelitis, retroperitoneal tumors.

COLUMBUS HOSPITAL

DANIEL A. ORTH—9. Operations.
H. E. DAVIS—9. Selective radiation therapy of malignancies; presentation of cases.
H. R. KENNY—10. Crohn's disease; presentation of cases.

COOK COUNTY HOSPITAL

RALPH C. SULLIVAN—9. Operative clinic.
MARSHALL DAVISON—10. Operative clinic: Thyroid.
RALPH B. BETTMAN—2. Operative clinic.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

RAYMOND W. MCNEALY—11. Lecture: Femoral hernia.
Staff—1. Demonstration on cadaver, surgery of the large bowel.

EVANGELICAL HOSPITAL

P. E. HOPKINS, G. E. JOHNSON, and JAMES PATEJDL—9. Operations.

HOLY CROSS HOSPITAL

V. F. TORCZYNSKI. Blood pressure and spinal anesthesia—study of 100 cases.
J. B. KARR. Injection treatment of hernia; herniotomy.
F. F. FRAIDER and N. B. PAVLETIC. Indirect inguinal hernia.
F. J. SALETTA. Thyroidectomy.
M. J. BADZMIEROWSKI. Thyroidectomy.
J. F. RUZIC and D. S. DICERO. Hernioplasty; vaginal hysterectomies; cholecystectomy; common duct exploration; thyroidectomy.

ILLINOIS MASONIC HOSPITAL

WILBUR E. KEESEY and J. WALTER JOHNSON—9. Cholecystectomy.
R. BRUCE MALCOLM—9. Partial gastrectomy.
JOHN R. HARGER and JOHN PISHOTTA—9. Cholecystduodenostomy.
C. DRUECK, SR. and HENRY E. OLIVER—9. Hemorrhoidectomy.
W. C. BORNEMEIER and I. KROSS—10. Herniotomy.
T. G. WALLIN and BEULAH WALLIN—10. Amputation of breast.
WARREN PUGH—10. Resection of colon.
JOHN F. DAVIS and A. E. WUESTEMAN—11. Colostomy.
PAUL MORF and EDWARD LEVISOHN—11. Appendectomy.
W. B. GERHARD and GEORGE GEYMER—11. Amputation of breast.

MERCY HOSPITAL

L. D. MOORHEAD—9. Operative and dry clinic: Thyroid.
A. K. VAUGHN—9. Treatment of varicose veins and ulcers.

MICHAEL REESE HOSPITAL

Staff—9. Operative clinic:
A. A. STRAUSS and S. F. STRAUSS. Gastro-intestinal.
M. L. PARKER. Subject to be announced.
JAMES PATEJDL. Stomach and colon.
S. L. GOLDBERG. Subject to be announced.
SAMUEL PERLOW. Peripheral vascular surgery.
Staff—9. Dry clinic: Thyroid symposium.
S. SOSKIN. Physiological disturbances in thyrotoxicosis.
ROBERT C. LEVY. Special tests in diagnosis of borderline thyroid cases.
O. SAPHIR and LEO M. ZIMMERMAN. Neoplasms of the thyroid.
M. L. PARKER and H. A. ROTH. Analysis of five years thyroid surgery and follow-up.
A. K. KOFF. Menstrual disturbances in relation to thyroid diseases.
Staff—2. Dry clinic:
S. L. GOLDBERG. Fish bone perforation of ileum.
M. L. PARKER. Periarteritis nodosa of bowel; foreign bodies in the stomach; regional ileitis.
R. B. BETTMAN and WILLIAM TANNENBAUM. Foreign body perforation of the intestinal tract; complications of gastro-enterostomy.
S. L. GOLDBERG and L. N. KATZ. Attempts at reducing experimental hypertension in dogs.

MUNICIPAL TUBERCULOSIS SANITARIUM

RICHARD DAVISON—9. Surgical treatment of tuberculosis.

NORWEGIAN-AMERICAN HOSPITAL

J. M. AMBERSON—9. Postoperative intravenous medication.
G. B. FAULEY—9:30. Surgical physiology of the thyroid.
A. M. JENSEN—9:30. Sulfanilamide in general surgery.
J. R. ORNDORFF—10. Technique of thyroidectomy.
F. H. FOWLER—10. Time question in appendicitis.
J. V. FOWLER, JR.—10:30. Pre- and postoperative care of the thyroid patient.

J. E. VERHAAG—10:30. Hernia from industrial causes.
M. M. CORBETT—10:30. Immediate treatment of abdominal injuries.
WARREN JOHNSON—11. Carcinoma of the stomach.
F. M. NICHOLSON—11. Treatment of scalp injuries.
M. E. LICHTENSTEIN—11:30. Technique of cholecystectomy.
J. V. FOWLER, SR.—11:30. Carcinoma of the breast.

PASSAVANT MEMORIAL HOSPITAL

JOHN A. WOLFER and associates—9. Tumor clinic.
FRANK QUEEN. The surgeon and pathologist as a diagnostic team, i.e., the decision for and conduct of biopsy with illustrative cases.
EARL E. BARTH. Important preliminaries prior to x-ray therapy.
H. E. DAVIS. Selection of tumor cases and x-ray vs. radium therapy.
HENRY JAFFE. Administration of radiation therapy for malignant tumors.
FRED MERRIFIELD. Our results with intra-oral tumors.
J. M. GREENE. Useful aids in differential diagnosis of tumors of the neck.
JOHN A. WOLFER. Our results with cancer of the breast.
E. M. SMITH, JR. Results with treatment of unusual cutaneous tumors.
HERMAN CHOR. The psychiatrist's part in a tumor clinic; illustrative cases.
EARL E. BARTH. Inspection of radiation therapy department of Northwestern University Medical School.

PRESBYTERIAN HOSPITAL

KELLOGG SPEED, CARL B. DAVIS, ALBERT H. MONTGOMERY, EDWIN M. MILLER, FRANCIS STRAUSS, HARRY OBERHELMAN, W. J. POTTS, HILLIER BAKER, FRANK V. THEIS, EGBERT H. FELL, R. K. GILCHRIST, and VERNON C. DAVID—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

CHARLES B. PUESTOW and staff—9. Operative and dry clinic:
CHARLES B. PUESTOW. Choledocostomy for stone; physiology of the common duct as related to surgery.
E. F. FOLEY. The jaundiced patient from the medical viewpoint.
WARREN H. COLE. Lesions of the cystic duct as related to gall-bladder disease.
LINDON SEED and J. KARABIN. Use of Vitamin K in hypoprothrombinemia.
CHARLES B. PUESTOW. Excision carcinoma of colon.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. LUKE'S HOSPITAL

GÉZA DE TAKATS and J. T. REYNOLDS—9. Operative clinic: Lumbar sympathectomy.
PAUL W. GREELEY—9. Operations: Construction of new eyebrows; full thickness skin graft; split skin graft.
H. E. MOCK and associates—9. Skull fracture exhibit.
JOHN S. COULTER—10. Physical therapy in peripheral vascular disease.
G. K. FENN—10. Extracardiac factors influencing coronary circulation.
G. W. SCUPHAM—10. Renal disease and hypertension.
R. B. CAPPS—11. Vascular reflexes during operations.
J. M. L. JENSEN—11. Paravertebral block of the sympathetics; technique and indications.
GÉZA DE TAKATS—11. Demonstration in vascular surgery: Cervical rib and aneurysm; wired aneurysm of aorta; congenital arteriovenous fistula of the hand; modified Kondoleon operation for thrombophlebitic induration.

ST. MARY OF NAZARETH HOSPITAL

T. M. LARKOWSKI—9. Operative clinic.
E. H. WARSZEWSKI—10. Operative clinic.
THEODORE STEINERT—10. Operative clinic.
G. M. MUELLER—11. Operative clinic.

Friday

ALBERT MERRITT BILLINGS HOSPITAL

ALEXANDER BRUNSCHWIG and WILLIAM E. ADAMS—9. Operative and dry clinic.

CHILDREN'S MEMORIAL HOSPITAL

LOUIS W. SCHULTZ—1. Operative clinic and demonstration of cases: Oral surgery.

COOK COUNTY HOSPITAL

LOUIS RIVER—9. Dry clinic: Abdominal incisions.
E. H. WARZEWSKI—10. Operative clinic: Reconstruction treatment of burns.
J. R. BUCHBINDER—1. Operative clinic: Gastric resection for duodenal ulcer.

Scientific Exhibit

Presented by following departments: Pathology, roentgenology, neurosurgery, blood bank, solutions laboratory, medical record library, therapeutics, and the Cook County Graduate School of Medicine.

MICHAEL REESE HOSPITAL

JAMES PATEJDL, A. A. STRAUSS, S. F. STRAUSS, M. L. PARKER, NATHAN N. CROHN, S. L. GOLDBERG, LEO M. ZIMMERMAN, and SAMUEL PERLOW—9. Operations.

MT. SINAI HOSPITAL

D. A. WILLIS—9. New type of hernioplasty.
E. I. GREENE—9. Operative clinic.
V. L. SCHRAGER and staff—9. Hernioplasty.
A. A. STRAUSS and S. STRAUSS—9. Surgery of the colon.
M. R. GUTTMAN—9. Plastic surgery of the nose.
JACOB LIFSCHUTZ—9. Endoscopy.
EMIL AISON—9. Oral surgery.
J. M. MORA—9. Thyroidectomy.

PASSAVANT MEMORIAL HOSPITAL

J. R. BUCHBINDER—9. Dry clinic: Illustrations of end-results in treatment of gastric and duodenal lesions.

PRESBYTERIAN HOSPITAL

FREDERICK B. MOOREHEAD—9. Plastic surgery.

RESEARCH AND EDUCATIONAL HOSPITALS

R. B. MALCOLM and staff—9. Operative and dry clinic:
R. B. MALCOLM. Radical resection for carcinoma of breast; surgical pathology of breast tumors; excision of branchial cleft cyst.
P. J. SARMA. Differential diagnosis of breast lesions.
T. J. WACHOWSKI. Radiation therapy of carcinoma of breast.
GEORGE DE TARNOWSKY. End-results in carcinoma of breast.

P. W. GREELEY—10. Plastic surgery, follow-up clinic.

Scientific Exhibit

Cancer of the large bowel and appendicitis.

ST. ELIZABETH'S HOSPITAL

Staff—9. Operative and dry clinics:
A. G. ZIMMERMAN. Thyroidectomy; treatise on pathology of the thyroid gland.
M. G. LUKEN. Hernia operations.
E. B. KALVELAGE. Surgery of the stomach.
Staff—1. Operative and dry clinics:
J. B. O'DONOGHUE. Complications in surgery of the gall-bladder and ducts.
J. P. WOITALEWICZ. Intestinal surgery.

ST. LUKE'S HOSPITAL

H. E. MOCK and associates—9. Skull fracture exhibit.

Days to be Announced

HOSPITAL OF ST. ANTHONY DE PADUA

F. B. OLENTINE and M. J. BADZMIEROWSKI. Thyroid operations.
J. J. SPRAFKA. Abdominal operations.
S. E. DONLON, H. P. SULLIVAN, FRED EHRMANN, W. H. BRADLEY, JOSEPH ZABOKRTSKY, and R. C. DRURY. Operative and dry clinic.
FRANK J. JIRKA. Lecture: Convulsions under anesthesia.

OBSTETRICS AND GYNECOLOGY

Monday

COOK COUNTY HOSPITAL

A. J. KOBAK and H. H. HILL—2. Studies in acriflavine and glycerine.
JAMES H. BLOOMFIELD—2. Demonstration of cases: Cesarean section and local anesthesia, or management of occiput posterior.
S. J. BENENSOHN—2. Lues in the prenatal clinic.
T. J. MORRIS—2. Puerperal infection.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

Tuesday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.
FRED L. ADAIR and staff—2. Dry clinic:
FRED L. ADAIR. Subject to be announced.
FREDERICK W. SCHULTZ. Management of birth trauma.
WILLIAM J. DIECKMANN. Treatment of the toxemias of pregnancy.
M. EDWARD DAVIS. Treatment of placenta previa.
GORDON T. BURNS. Treatment of abruptio placentae.
EDITH L. POTTER. A review of the post-mortem findings in 2,000 fetal and neonatal deaths.
CHARLOTTE L. CLANCY. Problems in the control of reproduction.

COOK COUNTY HOSPITAL

W. T. CARLISLE—9. Gynecological operations.
A. E. KANTER—10. Gynecological operations.

EVANSTON HOSPITAL

W. C. DANFORTH, R. M. GRIER, HOWARD J. HOLLOWAY, P. H. SMITH, and C. E. GALLOWAY—9. Operative clinic: Vaginal hysterectomy; abdominal hysterectomy.

MERCY HOSPITAL

HERBERT E. SCHMITZ and staff—9. Operative and dry clinic: Vaginal hysterectomy; Watkins transposition operation; Manchester-Fothergill operation for vaginal plastic; anterior colporrhaphy and perineorrhaphy; La Forte colpocleisis.
Staff—2. Demonstrations and discussions of obstetrical problems.

MICHAEL REESE HOSPITAL

J. L. BAER—9. Operation: Radical hysterectomy.
J. LACKNER—9. Operation: Total abdominal hysterectomy.
L. E. FRANKENTHAL—9. Operation: Cesarean section.

PASSAVANT MEMORIAL HOSPITAL

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Operative and dry clinic: Integration of newer anatomical studies with clinical gynecology.

RAVENSWOOD HOSPITAL

Staff—10. Demonstration of cases.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. JOSEPH HOSPITAL

CLYDE GEIGER—9. Operation for carcinoma of the cervix.

ST. MARY OF NAZARETH HOSPITAL

M. E. UZNANSKI—10. Operations.
A. S. SAMPOLINSKI—11. Vaginal hysterectomy.

WESLEY MEMORIAL HOSPITAL

MARK T. GOLDSTINE and associates—9. Operative and dry clinic: Gynecological.
WILLIAM B. SERBIN—9. Operative and dry clinic: Obstetrical.
GARWOOD C. RICHARDSON—10. Operative and dry clinic: Obstetrical.
CHARLES B. REED—11. Operative and dry clinic: Obstetrical.

Wednesday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.
FRED L. ADAIR and staff—2. Dry clinic:
H. CLOSE HESSELTINE. The use of sulfanilamide, sulfapyridine and sulfathiazole in obstetrics and gynecology.
ROBERT G. BLOCH. Tuberculosis in pregnancy.
FRANKLIN F. SNYDER. Treatment of asphyxia neonatorum.
HENRY T. RICKETTS. Diabetes in pregnancy.

SIMON L. WOLTERS. Treatment of carcinoma of the uterus.
JOHN H. MORTON. Clinical studies in uterine motility.
RUTH WATTS. Studies of ovarian tumors.

CHICAGO MEMORIAL HOSPITAL

HARRY B. W. BENARON, JAMES E. FITZGERALD, WILLIAM F. HEWITT, GEORGE N. SCHIFF, BEATRICE E. TUCKER, and HARRY L. MEYERS—9. Operative and dry clinic:
HARRY B. W. BENARON. Presacral block in obstetrics.
GEORGE N. SCHIFF. Obstetrical analgesia.
JAMES E. FITZGERALD. Effect of Vitamin K in labor.
BEATRICE E. TUCKER and HARRY L. MEYERS. Manchester anterior colporrhaphy and perineorrhaphy.

COOK COUNTY HOSPITAL

HERBERT E. SCHMITZ—9. Gynecological operations.
A. F. LASH—10. Gynecological operations.

EVANGELICAL HOSPITAL

P. ARTHUR DELANEY and associates. Clinical pathological conference, unusual uterine pathology: Endometrial sarcoma; malignancy (leiomyosarcoma) in uterine fibroids; sarcoma botryoides, child 2½ years old; adenomyoma uteri (Frankel), endometriosis; actinomycosis in ovaries; granulosa cell tumor of ovary (microscopic); carcinoma in cystic ovaries; carcinoma in solid ovaries; ectopic tubal pregnancy with tuberculous salpingitis.

LORETTO HOSPITAL

HARVEY LITTLE and NICHOLAS BALSAMO—9. Operative clinic: Gynecological.

MICHAEL REESE HOSPITAL

Staff—9. Dry clinic:
A. F. LASH. Early treatment of puerperal sepsis peritonitis.
H. STRAUSS and S. MESIROW. Ball method of cephalopelvimetry.
I. F. STEIN. Gynecography.
M. R. COHEN. Clinical use of pregneninolone.
FRANK RUBOVITS. Serum therapy in shock or hemorrhage.
E. J. DECOSTA. Further development of the photostethoscope.
J. LACKNER and A. TULSKY. Effect of prephysin on the ovary.

PRESBYTERIAN HOSPITAL

N. SPROAT HEANEY, AARON KANTER, EDWARD C. ALLEN, A. KLAWANS, FRED PRIEST, and HARRY BOYSEN—9. Operations: Gynecological.

RESEARCH AND EDUCATIONAL HOSPITALS

F. H. FALLS and staff—2. Operative and dry clinic:
W. H. BROWNE. Low cervical cesarean section.
J. R. WOLFF. Six cases of early carcinoma of cervix uteri.
R. A. LIFVENDAHL. Vaginal hysterectomy for prolapse uterus.
F. H. FALLS. Vaginal hysterectomy for fibroids.
H. H. HILL. Report of two cases of ovarian pregnancy.
A. F. LASH. Supracervical hysterectomy.

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. ANNE'S HOSPITAL

J. L. FLEMING—1. Dry clinic: Hematogenous streptococcus puerperal infection; chorionephitbelionia of fallopian tube; inversion of uterus.
R. J. HAWKINS—1. Essential delivery room equipment.

SWEDISH COVENANT HOSPITAL

R. A. LIFVENDAHL, G. F. HIBBERT, G. L. ROSENE, and H. J. TIMERMAN—9. Vaginal hysterectomy; cesarean section case studies.

WASHINGTON BOULEVARD HOSPITAL

PAUL C. FOX—9. Operative and dry clinic.

WOMEN AND CHILDREN'S HOSPITAL

AMELIA GIRYOTAS, ELOISE PARSONS, PEARL STETLER, MARY EDITH WILLIAMS, and MAUDE HALL WINNETT—9. Surgical and gynecological operations.
BEATRICE TUCKER—9. Operations.
BERTHA VAN HOOSEN—9. Demonstration: Teaching gynecology by models.

Thursday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.
FRED L. ADAIR and staff—2. Dry clinic:
WILLIAM J. DIECKMANN. Results of treatment of toxemia patients.
M. EDWARD DAVIS. Rational endocrine therapy in obstetrics and gynecology.
ALLAN T. KENYON. Metabolic influences of the steroid hormones.
MELBOURNE W. BOYNTON. Present-day management of the menopause.
KATSUJI KATO. Vitamin K deficiency in the newborn.
ALICE CHILDS. Heart disease in pregnancy.
DONALD M. SCHUITEMA. Subject to be announced.

COLUMBUS HOSPITAL

CHANNING BARRETT—9. Operations.

COOK COUNTY HOSPITAL

E. W. FISCHMANN—9. Gynecological operations.
J. P. GREENHILL—10. Gynecological operations.

GRANT HOSPITAL

FREDERICK H. FALLS—9. Gynecological operation.
E. W. FISCHMANN—2. Gynecological operation.

HOLY CROSS HOSPITAL

P. E. LAWLER. Manikin demonstration.
F. F. FRAIDER and N. B. PAVLETIC. Hysterectomy.
F. J. SALETTA. Hysterectomy.

ILLINOIS MASONIC HOSPITAL

F. O. BOWE and PAULINE LANGE—9. Cesarean section.
DANIEL W. JEFFRIES and R. LEVISOHN—9. Ventral fixation of uterus.
HAROLD W. MILLER and GLEN NELSON—10. Hysterectomy.
ALLAN H. FERGUSON—10. Perineorrhaphy.
A. J. SCHOENBERG and M. OWEN WILKINS—11. Amputation of cervix.

NORWEGIAN-AMERICAN HOSPITAL

B. W. BREISTER—9. Indications for cesarean section.
P. F. SNYDER—11. Differential diagnosis of uterine tumors.

PASSAVANT MEMORIAL HOSPITAL

ARTHUR H. CURTIS and GEORGE H. GARDNER—9. Operative and dry clinic: Vaginal plastic procedures and abdominal total hysterectomies.

DAVID S. HILLIS and associates—2. Dry clinic: Cephalopelvic disproportion.

RESEARCH AND EDUCATIONAL HOSPITALS

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. LUKE'S HOSPITAL

Staff—10. Operations.

Friday

CHICAGO LYING-IN HOSPITAL

FRED L. ADAIR, WILLIAM J. DIECKMANN, M. EDWARD DAVIS, H. CLOSE HESSELTINE, and FRANKLIN F. SNYDER—9. Operations.

FRED L. ADAIR and staff—2. Dry clinic:
- FRED L. ADAIR. Treatment of uterine prolapse.
- FRANKLIN F. SNYDER. Subject to be announced.
- H. CLOSE HESSELTINE. Management of chronic and resistant vaginal trichomoniasis.
- HILGER P. JENKINS. Acute appendicitis of pregnancy.
- LUCILE HAC. Transportation of gonococcal cultural material.
- PRICILLA OUDA. Vomiting as a complication of obstetrical anesthesia.

COOK COUNTY HOSPITAL

WILLIAM H. BROWNE—9. Gynecological operations.

FREDERICK H. FALLS—10. Gynecological operations.

AUGUST DARO. Use of epinephrine in acute inversion of the uterus.

LOUIS RUDOLPH. Ring dystocia constriction.

JAMES E. FITZGERALD. Heart disease in pregnancy.

DAVID S. HILLIS. Recognition of cephalopelvic disproportion.

AUGUSTA WEBSTER. Vitamin K in pregnancy.

MICHAEL REESE HOSPITAL

WILLIAM RUBOVITS—9. Neugebauer-LaForte operation for prolapse.

RALPH REIS—9. Vaginal hysterectomy.

I. F. STEIN—9. Bilateral ovarian resection.

M. L. LEVENTHAL—9. Manchester operation for prolapse of uterus.

MT. SINAI HOSPITAL

A. E. KANTER and LOUIS RUDOLPH. Gynecological operations.

A. F. LASH. Vaginal hysterectomy.

H. BUXBAUM, C. NEWBURGER, and A. H. GOLDFINE. Obstetrical clinic.

RESEARCH AND EDUCATIONAL HOSPITALS

F. H. FALLS and staff—2. Operative and dry clinic:
- G. H. REZEK. Supracervical hysterectomy.
- A. J. KOBAK. Report of three cases of interstitial ectopic pregnancy.
- W. H. BROWNE. Vulvectomy.
- R. A. LIFVENDAHL. LaForte operation for prolapse of uterus.
- V. C. FREDA. Skin test for pregnancy.
- F. H. FALLS. Vaginal hysterectomy under local anesthesia.

Scientific Exhibit

Essentials of prenatal care; puerperal sepsis; care of the premature infant; ectopic pregnancies.

ST. ELIZABETH'S HOSPITAL

B. S. MILTON—9. Hysterectomy for fibroid tumors; discussion of pathology of fibroid tumors.

J. K. NARAT—1. Tumors of the uterus.

J. R. LAVIERI and F. J. WALSH—3. Surgical complications in obstetrics.

Days to be Announced

HOSPITAL OF ST. ANTHONY DE PADUA

M. A. WEISSKOPF. Obstetrical operations.

GENITO-URINARY SURGERY

Tuesday

EVANSTON HOSPITAL

JAMES I. FARRELL—9. Infections of the urinary tract.

MICHAEL REESE HOSPITAL

Staff—9. Dry clinic:
- GUSTAV KOLISCHER. Carcinoma of breast successfully treated with macrophages.
- IRVIN S. KOLL. Massive renal calculi.
- HARRY C. ROLNICK. Radical perineal prostatectomy.
- A. E. JONES. Hydronephrosis due to retroperitoneal lymphosarcoma.
- IRVING SHAPIRO. Ureteral injuries in gynecological operations.
- FREDERICK LIEBERTHAL. Tumors of kidney.
- J. S. GROVE. Ureteral calculus in case of solitary kidney; urosepsis.

PRESBYTERIAN HOSPITAL

HERMAN L. KRETSCHMER, ROBERT HERBST, NORRIS J. HECKEL, and JAMES MERRICKS—9. Operative clinic and demonstration of cases.

RAVENSWOOD HOSPITAL

Staff—10. Demonstration of cases.

ST. JOSEPH HOSPITAL

Staff—9. Dry clinic.

WESLEY MEMORIAL HOSPITAL

VICTOR D. LESPINASSE—9. Operative and dry clinic.

DONALD K. HIBBS—10. Operative and dry clinic.

Wednesday

ALBERT MERRITT BILLINGS HOSPITAL

CHARLES B. HUGGINS—9. Operative and dry clinic.

HENROTIN HOSPITAL

C. O. MILLER—9. Neoplasm of the kidneys.

LORETTO HOSPITAL

CARL J. UTHOFF—9. Operative and dry clinic.

MERCY HOSPITAL

H. E. LANDES—9. Clinical cystometry, an aid in the diagnosis of disturbances of micturition.

MICHAEL REESE HOSPITAL

G. KOLISCHER, I. S. KOLL, J. S. EISENSTAEDT, H. C. ROLNICK, A. E. JONES, I. J. SHAPIRO, F. LIEBERTHAL, and J. Grove—9. Operations.

MUNICIPAL TUBERCULOSIS SANITARIUM

D. F. RUDNICK—9. Genito-urinary tuberculosis.

ST. ANNE'S HOSPITAL

H. J. DOOLEY and C. C. SAELHOF—9. Operations.
H. J. DOOLEY—1. Anesthesia in genito-urinary surgery.
C. C. SAELHOF—1. Surgery of prostate in diabetics, discussion and demonstration.

ST. BERNARD'S HOSPITAL

A. J. SULLIVAN—9. Prostatic operations.

SWEDISH COVENANT HOSPITAL

B. E. FILLIS, J. T. GERNON, and K. D. KOHLSTEDT—9. Genito-urinary problems.

VETERANS ADMINISTRATION FACILITY

T. G. MCDOUGALL—9. Operations.

Thursday

ALEXIAN BROTHERS' HOSPITAL

JULIUS M. GLASSER—9. Prostatectomy, 2 cases.
EDWARD F. HESS—9. Dry clinic: Nephrotomy; cystoscopy and pyelography.
EDWARD W. WHITE—10. Prostatic electroresection; ureteral stone.
A. J. WOCHINSKI—10. Prostatic electroresection; fulguration of bladder tumor, 2 cases.

CHICAGO MEMORIAL HOSPITAL

VINCENT J. O'CONOR, JOHN P. O'NEIL, J. WILLIAM PARKER, and KENNETH SOKOL—9. Operative and dry clinic.

CHILDREN'S MEMORIAL HOSPITAL

HERMAN L. KRETSCHMER—9. Operative clinic and demonstrations: Urological problems in infancy and childhood.

COLUMBUS HOSPITAL

WILLIAM H. GEHL—9. Transurethral prostatic resection.

GRANT HOSPITAL

EDWARD F. HESS—9. Demonstration of cases.
WILLIAM H. GEHL—9. Operations.

ILLINOIS MASONIC HOSPITAL

WILLIAM H. GEHL and F. L. CHENOWETH—9. Nephrectomy.
C. OTIS RITCH and O. F. HARRIS—9. Suprapubic cystotomy.
EDWARD W. WHITE and CHARLES MURRAY—10. Prostatectomy.
R. B. GAINES—10. Nephrolithotomy.
CLARENCE C. SAELHOF—11. Cystocopic examination.

MICHAEL REESE HOSPITAL

G. KOLISCHER, I. S. KOLL, J. S. EISENSTAEDT, H. ROLNICK, A. E. JONES, I. J. SHAPIRO, F. LIEBERTHAL, and J. S. GROVE—9. Operations.

NORWEGIAN-AMERICAN HOSPITAL

D. F. RUDNICK—10. Transurethral resection of prostate.

PASSAVANT MEMORIAL HOSPITAL

LEANDER W. RIBA—2. Dry clinic: Chronic female urethritis and its treatment.
L. L. VESEEN—3. Diagnosis and treatment of tumors of the testes.

ST. LUKE'S HOSPITAL

L. E. SCHMIDT and staff—9. Operative and dry clinic.

WASHINGTON BOULEVARD HOSPITAL

VINCENT J. O'CONOR—9. Operative and dry clinic.

Friday

MT. SINAI HOSPITAL

H. C. ROLNICK—9. Operations.

ST. ELIZABETH'S HOSPITAL

T. G. MCDOUGALL—3. Nephrectomy; discussion of tumors of the kidney.

Days to be Announced

COOK COUNTY HOSPITAL

HARRY CULVER, HARRY C. ROLNICK, D. F. RUDNICK, and L. L. VESEEN. Operations.

HOSPITAL OF ST. ANTHONY DE PADUA

OTTO JIRSA. Operative and dry clinic: Cystoscopy.

RESEARCH AND EDUCATIONAL HOSPITALS

C. M. MCKENNA, J. H. KIEFER and staff—9. Operative and dry clinic: Transurethral resection for hypertrophied prostate gland; suprapubic operation for hypertrophied prostate gland; nephrostomy and removal of aberrant vessels for hydronephrosis; repair of undescended testicle; total cystectomy following transplantation of ureters for bladder exstrophy; end-results of correction of hypospadias; end-results of repair of undescended testicle.
R. C. HERROLD—9. Evaluation of urinary antiseptics in treatment of infections.

FRACTURES AND OTHER TRAUMAS

Monday

COOK COUNTY HOSPITAL

WILLIAM R. CUBBINS, J. J. CALLAHAN and C. S. SCUDERI—1. Operative clinic.

Tuesday

CHICAGO MEMORIAL HOSPITAL

T. C. BROWNING, EDWARD L. COMPERE, ARTHUR H. CONLEY, EMILE C. DUVAL, C. R. G. FORRESTER, A. H. MASON, FRED M. MILLER, S. PERRY ROGERS, HORACE STIMSON, and GEORGE M. LANDAU—9. Operative and dry clinic:
C. R. G. FORRESTER. Shoulder fractures.
A. H. MASON. Fractures of the scaphoid.
FRED M. MILLER and T. C. BROWNING. Fractures of foot and ankle.
EDWARD L. COMPERE. Vitamin D and calcium in fracture repair.
S. PERRY ROGERS. Vitallium in fractures.
EMILE C. DUVAL. Internal semilunar cartilage as a factor in industrial surgery.
HORACE STIMSON. Treatment of fractures of spine.

EVANSTON HOSPITAL

MARCUS H. HOBART. Malunited Colles' fracture.
DWIGHT F. CLARK. Intramedullary splinting with cow's horn; four-year end-result.

RAVENSWOOD HOSPITAL

Staff—10. Demonstration of cases.

ST. JOSEPH HOSPITAL

HUGH MCKENNA—9. Demonstrations; Motion pictures and slides of fractures of the neck of femur and demonstration of a device used in securing x-ray films during the stage of reduction.

WASHINGTON BOULEVARD HOSPITAL

ARTHUR R. METZ—9. Operative and dry clinic.

Wednesday

CHILDREN'S MEMORIAL HOSPITAL

ALBERT H. MONTGOMERY, JAY IRELAND, and W. J. POTTS—2:30. Demonstration of cases: Fractures of forearm; supracondylar fractures of humerus; bone cysts; skull fractures.

COOK COUNTY HOSPITAL

GEORGE L. APFELBACH—2. Operative clinic: Flange operation for fractured neck of femur; open reduction of the forearm with intramedullary peg; reconstruction of malunited Colles' fracture.

HENROTIN HOSPITAL

RALPH A. KORDENAT—9. Operations.
JOHN A. GRAHAM—9. Demonstration, with lantern slides: Fracture of distal end of radius.
JOHN J. EICHSTADT—9. Dry clinic: Care of fractures.

JACKSON PARK HOSPITAL

F. G. MURPHY—9. Operations.

MERCY HOSPITAL

F. C. JACOBSEN—2. Industrial fractures.
JOHN D. CLARIDGE—2. Fractures of the humerus.
J. M. LEONARD—4. Forearm fractures.

PASSAVANT MEMORIAL HOSPITAL

PAUL B. MAGNUSON and JAMES K. STACK—3. Dry clinic: Joint débridement; cases illustrating end-results of treatment of traumatic arthritis.

ST. ANNE'S HOSPITAL

J. J. CALLAHAN—1. Dry clinic: Shoulder injuries; congenital dislocation of patella.
T. E. MEANY—1. Dry clinic: Fracture of femur in children—Paget's disease.

SWEDISH COVENANT HOSPITAL

O. T. ROBERG and O. T. ROBERG, JR.—9. Fractures.

Thursday

COLUMBUS HOSPITAL

GEORGE BEECHER—9. Dry clinic.

COOK COUNTY HOSPITAL

WILLIAM R. CUBBINS and JAMES J. CALLAHAN—1. Operations.

NORWEGIAN-AMERICAN HOSPITAL

H. A. SOFIELD—11. Steel pin fixation of fractures of neck of the femur.

Friday

COOK COUNTY HOSPITAL

COOK COUNTY GRADUATE SCHOOL OF MEDICINE

Staff—1. Demonstration on cadaver. Fractures and traumatic surgery.

PRESBYTERIAN HOSPITAL

Staff—9. Operative and dry clinic:

EDWIN M. MILLER. Improved methods in dealing with supracondylar fractures of the elbow; resection of tumor of metacarpal bone with free graft from tibia; x-rays, microscopic pathology and clinical result.
WILLIS J. POTTS. Improved method of immobilization of fractures of both bones of leg.
EGBERT FELL. Fractures of the capitellum of the humerus.
EDWARD L. COMPERE. Evaluation of methods of internal fixation of fractures of neck of the femur.
E. J. BERKHEISER. Dislocations of cervical vertebrae; arthrodesis of shoulder; tendon transplants.
KELLOGG SPEED. Reconstruction problems following trauma in hip region; illustrated talk on wounds of war; fractures about elbow.

Days to be Announced

HOSPITAL OF ST. ANTHONY DE PADUA

F. W. SLOBE. Operative clinic: Fractures; tendon sutures; care of wounds; treatment of burns.

ORTHOPEDIC SURGERY

Monday

CHILDREN'S MEMORIAL HOSPITAL

FREMONT A. CHANDLER, F. SEIDLER, C. N. PEASE, J. R. NORCROSS, and L. SMITH—2:30. Operative and dry clinic: Osteomyelitis; resection of ilium; rare infection of joint; tuberculous metatarsal and tuberculous ankle; patellar advancement; radial stripping for pronation deformity; unusual tumor of femur; unusual tumor of tibia; anterior dislocation of hip; torticollis, six cases; Erb's palsy; back knee; medial dislocation of patella; arachnodactylia, two cases; osteochondromatous of hip; overgrowth of spine.

RESEARCH AND EDUCATIONAL HOSPITALS

H. B. THOMAS, F. W. HARK, C. N. LAMBERT, and S. L. ODGERS—2. Operative and dry clinic: Fusion of tuberculous hip; end-results of tuberculosis of hip and spine; tendon transplant to shoulder; results of tendon transplant for brachial plexus injury to shoulder; use of shift casts in treatment of scoliosis.

Tuesday

CHILDREN'S MEMORIAL HOSPITAL

FREMONT A. CHANDLER, F. SEIDLER, C. N. PEASE, J. R. NORCROSS, and L. SMITH—9. Operative clinic: Extra-

peritoneal resection of obturator nerve, two cases; Lowman operation for flat feet; triple arthodesis; Chandler patellar advancement operation; spine fusion; torticollis, four to five weeks of age; hip fusion operation; popliteal neurectomy.

COOK COUNTY HOSPITAL

ARTHUR H. CONLEY and DONALD MILLER—9. Operations.
MARCUS H. HOBART and FELIX JANSEY—10. Operative clinic: Shoulder injuries.

EVANSTON HOSPITAL

R. C. LONERGAN—9. Operative clinic: Internal derangement of knee.

MICHAEL REESE HOSPITAL

PHILIP LEWIN, D. H. LEVINTHAL, SIDNEY SIDEMAN, IRVING WOLIN, C. N. PEASE, FRANK GLASSMAN, and J. G. FINDER—9. Operative clinic: Spinal fusion for scoliosis; Hoke arthrodesis for paralytic foot; laminectomy for intervertebral disc protrusion; poliomyelitis reconstruction; knee joint operation; hip joint operation.

ST. JOSEPH HOSPITAL

Staff—9. Demonstrations: Lantern slides and moving pictures of fractures.

SHRINERS' HOSPITAL FOR CRIPPLED CHILDREN

B. H. MOORE and H. A. SOFIELD—9. Operative clinic.

WESLEY MEMORIAL HOSPITAL

FELIX M. JANSEY—9. Operative and dry clinic.
PHILIP H. KREUSCHER—10. Operative and dry clinic.
HAMPER KELIKIAN—11. Operative and dry clinic.
O. H. HORRALL—11. Operative and dry clinic.

Wednesday

COOK COUNTY HOSPITAL

PHILIP LEWIN and SIDNEY SIDEMAN—9. Operative clinic.
DANIEL H. LEVINTHAL and staff—9. Dry clinic.
DANIEL H. LEVINTHAL—2. Operative clinic. Derangement of the knee joint; spinal fusion; arthroplasty of hip using vitallium cup; arthrodesis of foot for poliomyelitis.

LORETTO HOSPITAL

BEVERIDGE MOORE, JAMES A. VALENTINE, and FRED CARLS—10. Operative and dry clinic.

MICHAEL REESE HOSPITAL

Staff—2. Dry clinic: Dunlop traction treatment of transcondylar fractures of the elbow in children; Risser method in scoliosis; club feet; Legg-Calve-Perthes lesion; tuberculosis of the hip joint; tuberculosis of the spine; chronic osteomylitis; rickitic deformities; postoperative laminectomy for disc protrusion; open reduction of congenital hip dislocation; flexorplasty of thumb; spina bifida with paraplegia; vitallium cup hip arthroplasty; leg equalization by epiphyseal arrests; leg shortening.

PASSAVANT MEMORIAL HOSPITAL

PAUL B. MAGNUSON and JAMES K. STACK—3. Dry clinic: Joint debridement; cases illustrating end-results of treatment of traumatic arthritis.

ST. ANNE'S HOSPITAL

J. J. CALLAHAN and R. E. MEANY—9. Operations.

ST. LUKE'S HOSPITAL

HENRY B. THOMAS—10. Bone changes in connection with thyroid disease.
FRED W. HARK—10. Bone changes in connection with sickle-cell anemia.
CLAUDE N. LAMBERT—10. Bone tumors.
H. E. MOCK, ALVIN R. MORROW, CHARLES E. SHANNON, JOHN L. LINDQUIST, and DONNELL C. HOWE—11. Dry clinic: Conservative treatment of fractures of neck of femur; fractures of fingers and toes; supracondylar fractures of humerus; malunited fractures of ankle.
JOHN W. ELLIS—11. Compression fractures of vertebrae using Ryerson hyperextension device.
EDWIN W. RYERSON—1:30. Dry clinic: Shelf operation at hip; reconstruction of hip joint; osteotomy to produce back-knee.
FREMONT W. CHANDLER—2. Operative clinic: Aseptic necrosis of head of femur.
ROBERT E. RITTER—2:30. Arthroplasty.
HAROLD A. SOFIELD—3. Discussion of leg lengthening operations; indications and technique.
Open forum—3:30.

SHRINERS' HOSPITAL FOR CRIPPLED CHILDREN

Staff—3. Clubfoot clinic.

VETERANS ADMINISTRATION FACILITY

S. K. LIVINGSTON—10. Dry clinic: Peripheral vascular diseases.

Thursday

ALEXIAN BROTHERS' HOSPITAL

GEORGE L. APPELBACH—10. Demonstration of examination for lower back pain; non-union of neck of femur.

ALBERT MERRITT BILLINGS HOSPITAL

CLY H. HATCHER and DALLAS B. PHEMISTER—9. Operative and dry clinic.

COLUMBUS HOSPITAL

FREDERICK MUELLER—9:30. Dry clinic.

COOK COUNTY HOSPITAL

FRANK G. MURPHY and WALTER FISCHER—9. Operations.
E. J. BERKHEISER—10. Operations.
ARTHUR H. CONLEY and staff—10. Dry clinic.

GRANT HOSPITAL

JOSEPH E. ALLEGRETTI—2. Treatment of arthritis.
GEORGE L. APFELBACH—2. Demonstration of examination for lower back pain; open reduction of fracture.

HOLY CROSS HOSPITAL

C. P. GALANTI. Rare bone tumors.

ILLINOIS MASONIC HOSPITAL

CHARLES N. PEASE—9. Fixation of hip.
WALTER R. FISCHER—10. Osteomyelitis.

MICHAEL REESE HOSPITAL

PHILIP LEWIN, D. H. LEVINTHAL, SIDNEY SIDEMAN, IRVING WOLIN, C. N. PEASE, FRANK GLASSMAN, and J. G. FINDER—9. Operative clinic: Spinal fusion for scoliosis; Hoke arthodesis for paralytic foot; laminectomy for intervertebral disc protrusion; poliomyelitis reconstruction; knee joint operation; hip joint operation.

RESEARCH AND EDUCATIONAL HOSPITALS

H. B. THOMAS, F. W. HARK, C. N. LAMBERT, and S. L. ODGERS—2. Operative and dry clinic: Open reduction for slipped epiphysis of the femur and insertion of fibular graft through neck; end-results of hip joint surgery, particularly fractures and epiphyseal separations.

SHRINERS' HOSPITAL FOR CRIPPLED CHILDREN

B. H. Moore and H. A. Sofield—9. Operative clinic.

Friday

COOK COUNTY HOSPITAL

Fred Hark and Claude Lambert—9. Operations.
H. Kelikian and Graham A. Kernwein—10. Operations.

MT. SINAI HOSPITAL

C. L. Jacobs and Leo F. Miller—9. Operations.

PASSAVANT MEMORIAL HOSPITAL

Emil Hauser—10. Case presentations: Fusion of hip for tuberculosis; spinal fusion, presenting an unusual complication; Brodie's abscess, illustrating results of chemotherapy; after-result of treatment of delayed union in fracture of humerus; after-treatment for fracture of calcaneum.

ST. MARY OF NAZARETH HOSPITAL

L. M. Czaja—9. Operations.

THORACIC SURGERY

Tuesday

COOK COUNTY HOSPITAL

Jerome R. Head—10. Dry clinic.
R. B. Bettman—2. Operative clinic.

EVANSTON HOSPITAL

Jerome R. Head—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

Willard Van Hazel—9. Operative and dry clinic: Thoracoplasty; thoracotomy for chronic empyema; discussion of surgical drainage of lung abscess; lobectomy for bronchiectasis.

Wednesday

VETERANS ADMINISTRATION FACILITY

Jerome R. Head—11. Dry clinic: Thoracoplasty.

Thursday

ALEXIAN BROTHERS' HOSPITAL

Minas Joannides and Louis J. Miller—9. Operative clinic: First-stage thoracoplasty; extrapleural pneumolysis, axillary approach; lobectomy for bronchiectasis. Dry clinic: Hedblom's syndrome, clinical and radiological aspects; chronic suppuration of the lung in relation to carcinoma of the lung.

ILLINOIS MASONIC HOSPITAL

Minas Joannides and L. J. Miller—2. Extrapleural pneumolysis.

MICHAEL REESE HOSPITAL

S. L. Goldberg—2. Stab wound of pericardium and lung.
Nathan N. Crohn—2. Concepts in treatment of empyema.
Max Biesenthal—2. A ten-year review of collapse therapy in treatment of tuberculosis.
R. B. Bettman and William Tannenbaum—2. Surgical approach to thoracic esophagus.
R. B. Bettman—2. Dry clinic: Interesting cases in thoracic surgery.
Dr. Lennons—2. Anesthesia in thoracic surgery.

Friday

MICHAEL REESE HOSPITAL

R. B. Bettman and William Tannenbaum—9. Operations.

MUNICIPAL TUBERCULOSIS SANITARIUM

Minas Joannides—9. Collapse therapy clinic.

PRESBYTERIAN HOSPITAL

John M. Dorsey—9. Operations.

NEUROSURGERY

Tuesday

ST. LUKE'S HOSPITAL

Eric Oldberg and Wesley A. Gustafson—9. Operations and demonstrations.

Wednesday

COOK COUNTY HOSPITAL

Harold C. Voris—9. Dry clinic: Operative treatment of head injuries.
Adrien Verbrugghen and associates—1. Operations.

PASSAVANT MEMORIAL HOSPITAL

Loyal Davis and John Martin—2. Dry clinic: Discussion of results of surgical treatment of essential hypertension; discussion of tumors of the nasopharynx, producing intracranial symptoms.

Thursday

COOK COUNTY HOSPITAL

Adrien Verbrugghen and associates—10. Dry clinic: Concussion of the brain.

MERCY HOSPITAL

Harold C. Voris—9. Dry clinic: Protruding intervertebral disc.

MICHAEL REESE HOSPITAL

Adrien Verbrugghen—2. Operative and dry clinic.

Friday

PRESBYTERIAN HOSPITAL

Adrien Verbrugghen—9. Operations.

ST. LUKE'S HOSPITAL

Eric Oldberg and Wesley A. Gustafson—9. Operations and demonstrations.

Days to Be Announced

RESEARCH AND EDUCATIONAL HOSPITALS

Eric Oldberg, Percival Bailey, Wesley A. Gustafson, and Milton Tinsley—9. Operations and demonstrations.

OPHTHALMOLOGY

Tuesday

CHICAGO MEMORIAL HOSPITAL

FRANCIS M. CRAGE, HERMAN P. DAVIDSON, and GLENWAY NETHERCUT—2. Operative and dry clinic.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

EVANSTON HOSPITAL

G. R. SOPER, GLENN J. GREENWOOD, and R. H. HENDERSON—9. Operations.

GRANT HOSPITAL

OSCAR H. KRAFT—9. Operative and dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

THOMAS D. ALLEN, V. M. LEECH, and G. W. NETHERCUT—2. Operative clinic: Cataracts; ptosis; glaucoma.

PASSAVANT MEMORIAL HOSPITAL

SANFORD GIFFORD and associates—9. Dry clinic: Retina detachment; retinoplasty; plastic surgery of the eyelids.

RESEARCH AND EDUCATIONAL HOSPITALS

H. J. SMITH—9. Management of foreign bodies.
HALLARD BEARD—9. Surgical treatment of glaucoma.

ST. JOSEPH HOSPITAL

Staff—9. Operative and dry clinic.

ST. LUKE'S HOSPITAL

ELMER VORISEK—2. Demonstration of cases.

WESLEY MEMORIAL HOSPITAL

PHILIP O'CONNOR—9. Operative and dry clinic.
WILLIAM A. MANN and IRVING PUNTENNEY—10. Operative and dry clinic.

Wednesday

CHILDREN'S MEMORIAL HOSPITAL

RICHARD GAMBLE and associates—4. Dry clinic.

COOK COUNTY HOSPITAL

JAMES E. LEBENSOHN and WILLIAM F. MONCREIFF—9. Operations.

ILLINOIS EYE AND EAR INFIRMARY

PETER C. KRONFELD—9. Gonioscopy.
KATHARINE CHAPMAN—9. Orthoptic clinic.
ROBERT VON DER HEYDT—3. Slit lamp demonstration.

MERCY HOSPITAL

CARL F. SCHAUB and L. G. HOFFMAN—9. Operative and dry clinic. Demonstration of verified cases of opticochiasmatic arachnoiditis.

ST. BERNARD'S HOSPITAL

C. P. SULLIVAN. Cataract operations.

SWEDISH COVENANT HOSPITAL

R. A. DAVIS—9. Operative and dry clinic: Glaucoma.

Thursday

COLUMBUS HOSPITAL

MICHAEL GOLDENBURG—9. Operations.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

ILLINOIS EYE AND EAR INFIRMARY

LOUIS G. HOFFMAN—9. Operative and dry clinic: Verhoeff suture.
E. K. FINDLAY—10. Dry clinic.
E. K. FINDLAY and Dr. CORBOY—2. Operative clinic.

ST. LUKE'S HOSPITAL

E. V. L. BROWN—9. Demonstration of cases.
FRANK BRAWLEY and JAMES W. CLARK—2. Demonstration of cases.

Friday

COOK COUNTY HOSPITAL

SANFORD R. GIFFORD—9. Operations.
EDWARD A. ROLING—10. Operations.

ILLINOIS EYE AND EAR INFIRMARY

PETER C. KRONFELD—9. Gonioscopy.
KATHARINE CHAPMAN—9. Orthoptics.
SAMUEL J. MEYER—2. Operative clinic: Cataracts; glaucoma.

RESEARCH AND EDUCATIONAL HOSPITALS

CARL APPLE—9. Recession operation for strabismus.
J. W. CLARK—9. Intracapsular extraction of cataract.

ST. ELIZABETH'S HOSPITAL

F. A. DULAK and OSCAR CLEFF—3. Operative and dry clinic: Glaucoma.

ST. LUKE'S HOSPITAL

RICHARD GAMBLE—3. Demonstration of cases.

OTOLARYNGOLOGY

Monday

ILLINOIS EYE AND EAR INFIRMARY

SAMUEL SALINGER—2. Operative and dry clinic: Plastic surgery about the nose.

Tuesday

ALBERT MERRITT BILLINGS HOSPITAL

JOHN R. LINDSAY—2. Exhibit and demonstration: Suppurations of the petrous pyramid; anatomical, pathological, and roentgenological considerations.

CHICAGO MEMORIAL HOSPITAL

OSCAR CLEFF, ALFRED LEWY, ROBERT LEWY, JAMES B. MCBEAN, GORDON H. SCOTT, and RICHARD W. WATKINS—2. Operations.

COOK COUNTY HOSPITAL

JACOB LIFSCHUTZ—2. Operative and dry clinic.
RAYMOND W. KERWIN—2. Operative and dry clinic.

EVANGELICAL HOSPITAL

G. HENRY MUNDT, J. R. SMITH, EVAN A. MILLER, and J. H. F. O'NEIL—9. Operations.

EVANSTON HOSPITAL

T. C. Galloway, H. C. Ballenger, and L. J. Lawson. Operations.
T. C. Galloway. Anerobic bone infections about the head.

ILLINOIS EYE AND EAR INFIRMARY

T. P. O'Connor—9. Plastic surgery, demonstration of patients, lantern slides, etc.
Carl Cristoph—9. Operative clinic: Radical mastoid.
E. J. Blonder—2. Labyrinthine diagnosis and demonstration of the galvanic following reaction.
M. A. Glatt—2. Operative clinic: Radical mastoid and external frontal.

RESEARCH AND EDUCATIONAL HOSPITALS

P. H. Holinger and A. H. Andrews, Jr.—9. Bronchoscopies.
F. L. Lederer and staff—2. Otorhinolaryngological operations.

ST. JOSEPH HOSPITAL

Staff—9. Operative and dry clinic.

WESLEY MEMORIAL HOSPITAL

T. P. O'Connor and Leonard C. DeLozier—10. Operative and dry clinic.
Tai Tong Ching—11. Operative and dry clinic.

Wednesday

COOK COUNTY HOSPITAL

S. J. Pearlman—10. Operative and dry clinic.
Jacob Lifschutz—2. Operative and dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

Dr. Woodruff. Laryngeal sinus and ear cases; endoral mastoid and sinus surgery.
T. P. O'Connor—2. Operative clinic.

JACKSON PARK HOSPITAL

Noah Fox—9. Operations.

PASSAVANT MEMORIAL HOSPITAL

John F. Delph and associates—9. Dry clinic: Laryngoscopy, bronchoscopy, esophagoscopy; presentation of exhibits and cases; carcinoma of larynx.

ST. ANNE'S HOSPITAL

J. W. Hayden—9. Operations.

ST. MARY OF NAZARETH HOSPITAL

F. J. Piszkiewics—9. Otological operations.

VETERANS ADMINISTRATION FACILITY

Max Cutler—11. Dry clinic: Carcinoma of the mouth and larynx. Problems in diagnosis and treatment; indications for surgery and irradiation in laryngeal carcinoma; presentation of cases.

Thursday

ALBERT MERRITT BILLINGS HOSPITAL

John R. Lindsay—2. Exhibit and demonstration: Suppurations of the petrous pyramid; anatomical, pathological, and roentgenological considerations.

COOK COUNTY HOSPITAL

Raymond W. Kerwin—2. Operative and dry clinic.

EVANGELICAL HOSPITAL

G. Henry Mundt, J. R. Smith, Evan A. Miller, and J. H. F. O'Neil—9. Operations.

GRANT HOSPITAL

Carroll W. Stuart—2. Oral surgery.
S. H. Soboroff—2. Nose and throat operation.

ILLINOIS EYE AND EAR INFIRMARY

E. J. Blonder—9. Operative and dry clinic: Radical mastoid.
M. A. Glatt—2. Osteomyelitis of the frontal bone.

ILLINOIS MASONIC HOSPITAL

Alva Sowers—9. Tonsillectomy.
Arthur Geiger—9. Deviated septum.
Maurice H. Cottle and Milton Levisohn—10. Mastoidectomy.
Herbert E. Taylor—11. Drainage maxillary sinus.
John Gilmore and Perry J. Melnick—12. Diagnosis and treatment of carcinoma.

MERCY HOSPITAL

Peter P. Ledermann—9. Disturbances of equilibrium.
Carl H. Christoph—9. Laryngeal malignancies.
Herbert Nash—9. Endaural operations.
G. T. Jordan—9. Anatomy of the temporal bone.

MUNICIPAL TUBERCULOSIS SANITARIUM

George Holmes—2. Bronchoscopy in tuberculosis.

NORWEGIAN-AMERICAN HOSPITAL

M. A. Glatt—9. Control of hemorrhage in tonsillectomy.
J. W. Harned—9:30. Danger signals in mastoid infection.

ST. LUKE'S HOSPITAL

Paul H. Holinger and Albert H. Andrews, Jr.—2. Peroral endoscopy; clinical presentation of bronchial and esophageal cases.
Horace R. Lyons, Walter H. Theobald and staff—3. Operative and dry clinic: Sulfanilamide therapy in acute mastoiditis and acute otitis media; Proetz treatment for sinusitis.

Friday

CHILDREN'S MEMORIAL HOSPITAL

Paul H. Holinger and Albert H. Andrews—10:30. Bronchoscopic clinic.

COOK COUNTY HOSPITAL

Jacob Lifschutz, T. C. Galloway, and R. W. Kerwin—10. Operative and dry clinic.

ILLINOIS EYE AND EAR INFIRMARY

Alfred Lewy—2. Operative clinic.

MT. SINAI HOSPITAL

S. M. Morwitz—9. Operations.

RESEARCH AND EDUCATIONAL HOSPITALS

L. W. Schultz—9. Operative and dry clinic: Cleft palate operation; presentation of interesting cases in oral surgery; stabilization of joints by injecting sclerosing agent; bilateral cleft lip operation; bilateral temporomandibular dislocation for 2½ months, reduction, end-results with motion pictures; vitalium cup arthroplasty of temporomandibular joint; presentation of case.

ST. ELIZABETH'S HOSPITAL

J. G. Weber and T. V. Carelli—3. Operative and dry clinic: Complications in mastoid surgery.